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OF THE

American Veterinary Medical Association

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CANINE DISTEMPER

A great deal of publicity has already been given to the results accomplished thus far in the investigation of canine distemper by the British scientists working under the auspices of the Field Distemper Research Committee. Briefly, it appears that a means has been discovered whereby dogs may be rendered solidly immune to distemper. Persons interested in dogs, in both England and the United States, have contributed the funds that have made the research work possible. A laboratory was erected and equipped at Mill Hill, near London, and the studies on canine distemper have been conducted by Dr. P. P. Laidlaw and Major G. W. Dunkin. Reports of their work have already appeared in British veterinary journals and in lay publications.

The work of Laidlaw and Dunkin has definitely confirmed the previous findings of Carré that the primary causative factor in canine distemper is a filtrable virus and not *Bacillus bronchisepticus*, as claimed by other investigators. They have found further that dogs can be rendered immune by the injection of a small dose of spleen extract (taken from infected animals) that has been treated with formalin, followed by a small dose of active virus, about ten days later, provided that these products are prepared in accordance with the standards which they have laid down. The security of the method has been demonstrated in England by the vaccination of more than 2,000 animals, which,

though later exposed to distemper infection, have proved to be immune.

No discovery of greater importance to dog-owners and veterinarians has ever been announced and there is already a keen desire on the part of many veterinarians to obtain this new immunizing treatment. Although it has been proven practical experimentally, and on a small scale, where all factors could be readily controlled, there are still many problems to be solved before the product can be satisfactorily manufactured and distributed on a commercial scale.

Dr. John Reichel, chairman of the A. V. M. A. Committee on Distemper, recently advised us to the effect that Dr. H. K. Wright, of the Veterinary Department of H. K. Mulford Company, was en route to England for the purpose of obtaining first-hand information and all details concerning the preparation of the new canine distemper immunizing agent, direct from the British scientists. In addition to representing the Mulford Laboratories, Dr. Wright has been commissioned as a special representative of the A. V. M. A. Committee on Distemper, and carried letters of introduction from Dr. John R. Mohler, of the U. S. Bureau of Animal Industry, and Mr. Chas. H. Tyler, secretary-treasurer of the American Distemper Committee. Undoubtedly it is safe to predict that, when the A. V. M. A. Committee on Distemper reports at the Detroit meeting, we will have some first-hand information on the very latest developments in connection with canine distemper.

It is our understanding that the U. S. Bureau of Animal Industry has as yet issued no licenses for the manufacture and sale of the Laidlaw-Dunkin product in this country. As a matter of fact, the new treatment is not yet available commercially even in England. No assurances can be given as to when the new product will be available. There are a great many details to be worked out in connection with producing and distributing the product commercially. One of the greatest difficulties is the maintenance of the virulence of the virus used for the second injection. It is stated that the virus loses its virulence very easily, unless kept very cold or in a frozen condition.

If you remember the last meeting of the A. V. M. A. in Detroit, you will not be found absent from the Dynamic City, August 13-16.

FIFTIETH ANNIVERSARY

The fiftieth anniversary of the founding of the Division of Veterinary Medicine, Iowa State College, will be celebrated at Ames, during Commencement, June 8, 9 and 10, 1929. The ceremonies will be inaugurated with an informal luncheon served to veterinary alumni and friends in the Memorial Union at 12:30 p. m. on Saturday, June 8. Dean C. H. Stange will preside at a meeting to be held in the Agricultural Assembly at three o'clock. The speakers will include Dr. R. M. Hughes, president of Iowa State College, Dr. J. H. McNeil, state veterinarian of New Jersey and former dean of the Veterinary Division at Iowa State College, and Dr. David S. White, Dean, College of Veterinary Medicine, Ohio State University.

At six o'clock, dinner will be served in the Memorial Union to the senior students, faculty and alumni. Dr. George C. Faville ('80), of Hampton, Va., a member of the first class graduated in veterinary medicine at Iowa State College, will represent the Veterinary Division on the banquet program. The banquet will be followed by an informal get-together of "vet. grads," old and new, with their wives and friends.

This occasion will mark the first time in the history of veterinary education in the United States that a veterinary college has had the privilege of celebrating the completion of fifty years of continuous existence. It will be at least five years before an opportunity will be presented for any other veterinary college in the United States to mark its fiftieth anniversary. Veterinary courses at the University of Pennsylvania and Ohio State University were inaugurated in 1884.

Detroit, the industrial hub of America.

INDEX NUMBER

This issue of the JOURNAL concludes Volume lxxiv (new series 27) and will be the last of the seven-number volumes. Hereafter there will be two volumes per year, of six numbers each, and the volumes will begin with the January and July issues. As a result of this change, we expect to avoid much of the confusion which has existed in the past, both in connection with subscriptions as well as references. Heretofore every other volume has been issued in parts of two calendar years, three months in one and three months in the other. Under the new plan, this will no longer be the case.

EXECUTIVE BOARD ELECTION

The election of a member of the Executive Board to represent District No. 4 is creating unusual interest in the states comprising the District. Where there seemed to be general apathy during the primaries, just the opposite condition now exists and as a result a very heavy vote is being polled. With the withdrawal of Dr. John R. Mohler from the race, only four candidates remain in the field and the friends and supporters of these four well-known veterinarians are whooping things up wherever there are any votes to be obtained. Approximately 250 ballots have already been received, a large vote for District No. 4, which is the smallest numerically of the five Executive Board districts in the United States. Cuba and South America are assigned to this District.

A member recently asked what would be done in the event of a tie vote in an Executive Board election. This contingency is covered by Section 1 of Article 10 of the By-laws. It is provided that in case of a tie vote the President shall call a meeting of the members from the Executive Board district concerned, who are in attendance at the annual meeting, and these members shall ballot for a choice between the tied nominees only. This is a situation that has never arisen, but it could happen easily enough. A tie vote in an Executive Board election might prove to be quite an incentive for members of the District to attend the next annual convention of the Association.

The polls will close on June 13.

Michigan, America's summer playground.

AN INVITATION FROM HUNGARY

During his trip to Europe, early in the year, Dr. Adolph Eichhorn, Director, Veterinary Department, Lederle Antitoxin Laboratories, had the pleasure of attending the ceremonies commemorating the completion of forty years of teaching by Professor Franz Hutyra, at the Royal Veterinary High School, Budapest, Hungary. Dr. Eichhorn reports that Hungarian veterinarians are looking forward with much pleasure to the visit of American veterinarians to Europe in 1930, on the occasion of the coming International Veterinary Congress. Through Dr. Eichhorn, our colleagues in Hungary have extended a very cordial invitation for Americans to pay them a visit. Their unbounded hospitality awaits us.

PROGRAM COMMITTEE MEETS

The A. V. M. A. Committee on Program met in Detroit on May 8 and made very substantial progress in the plans for the literary program for the Detroit meeting. Dr. E. P. Althouse, of Sunbury, Pa., secretary of the Section on General Practice, was unable to be present and sent his understudy, Dr. B. Scott Fritz, of Harrisburg, Pa. The Section on Sanitary Science and Food Hygiene was represented by the Secretary, Dr. F. H. Brown, state veterinarian of Indiana. Two members of the veterinary faculty at Cornell University are on the Committee, Dr. H. J. Milks, secretary of the Section on Small Animals, and Dr. W. A. Hagan, secretary of the Section on Education and Research. The new Section on Poultry was represented for the first time by Dr. R. O. Biltz, of Harrisburg, Pa.

The Committee on Program is now an entirely ex-officio committee, being composed of the secretaries of the various sections and the Secretary of the Association, who acts as chairman. The meeting lasted all day and at the afternoon session preliminary plans for the clinic were discussed. At this time the group was joined by Dr. E. E. Patterson, of Detroit, and Dr. B. J. Killham, of Lansing, members of the Local Committee on Arrangements. Dr. Patterson will have charge of the program for the clinic which, according to present plans, will be divided into five sections: cattle, horses, sheep and swine, small animals, and poultry. The clinic will be the only event scheduled on the program for Friday, August 16, and will be held at the State Fair Grounds.

We are planning to publish an outline of the literary program in the July issue of the JOURNAL.

Michigan is washed by all the Great Lakes but one.

EIGHT MORE STATES

This month we present figures on a small group of states which make up the middle class. These states have an A. V. M. A. membership strength varying from 35 to 52 members. As a matter of fact, two states included this month (Virginia and North Dakota) should have been considered last month. The truth of the matter is we had failed to take into consideration the large number of new members from North Dakota added to the roll since the report for 1928 was compiled. The new members in question filed their applications at the meeting in Minneapolis

last summer. These applications were given first listing in the September issue of the JOURNAL and were not completed until November 1, 1928. In the case of Virginia, somewhat the same condition holds, although the new members have been added more recently. In continuing our statistical review, we will adhere to the plan of making comparisons between the membership strength of 1923 and the present year, giving each state credit for all applications now pending, as in the case of the other states already considered.

Virginia shows up with a net increase of three members. North Dakota shows a gain of eleven members, a large part of this gain having been made last year, when we met in the Northwest. North Carolina has 44 members, a decrease of one from the year 1923. This change would have been in the other direction had it not been for the fact that the present year marked the completion of tuberculosis eradication work in North Carolina. When this work was brought to a close, a number of veterinarians in the state and federal service, who had been engaged in tuberculin testing, were transferred to other states and, as most of these men were members of the A. V. M. A., it resulted in quite a little lost ground for North Carolina as far as A. V. M. A. membership strength is concerned. Incidentally the states to which these men were transferred were the gainers.

The present membership strength in Connecticut is 44, which is a substantial increase over 1923, when the Nutmeg State had 34 members to her credit. Next comes Oregon with 42 members, an increase of five over 1923. Of the eight states being considered this month, Oregon is the only one that has had an A. V. M. A. convention during the period under consideration. The A. V. M. A. met at Portland in 1925, but that year did not see any great increase in Oregon's membership, mainly for the reason that the State had been pretty thoroughly worked during the years preceding the convention. It is believed, however, that Oregon is considerably above the average in the matter of the percentage of eligible veterinarians in the State who are members of the A. V. M. A.

From Oregon we take a great big jump to Florida. The Peninsula State has had a phenomenal increase in membership during the past few years, largely the direct result of a big increase in the number of veterinarians in the State. It is quite likely that some of the losses which have been charged to other states have been indirectly the result of Florida's increase. It will be

noted that the number of members has almost doubled, jumping from 22, in 1923, to 38 at the present time.

Oklahoma shows the greatest loss among the states covered this month. In 1923, Oklahoma was credited with 56 members. This number has been slowly but surely reduced to 37 at the present time. Agricultural conditions in Oklahoma have been none too good during recent years and this has indirectly had its effect upon the number of veterinarians in the State. Alabama is another state that shows a loss, the number of A. V. M. A. members having dropped from 44, in 1923, to 35 at the present time. This number is entirely too small, especially in view of the fact that Alabama, according to the latest figures, has 98 approved veterinarians. By way of comparison, Florida has 59 approved veterinarians and 38 A. V. M. A. members.

State	*1923*	*1929*	*Change*
Virginia	49	52	+ 3
North Dakota	38	49	+11
Connecticut	34	44	+10
North Carolina	44	43	— 1
Oregon	37	42	+ 5
Florida	22	38	+16
Oklahoma	56	37	—19
Alabama	44	35	— 9

There are thirteen states more to be covered. These range in membership strength from thirty down to a dozen.

The A. V. M. A. meeting in Detroit August 13-16, will be truly international—Canadian-American

STILL CLIMBING

Membership figures in the A. V. M. A. are still climbing. Compare the following data on applications with those published in May:

	1925	1926	1927	1928	1929
January	2	4	10	15	42
February	9	22	28	36	45
March	6	10	14	41	24
April	10	12	15	57	26
May	7	28	16	26	56
June	11	19	12	34	135
Totals	45	95	95	209	328

It will be noted that the 135 applications listed this month bring the total for the first six months of 1929 up to 328. The grand total of applications filed since the Minneapolis meeting is now 394, just eighteen less than the number for the full year 1928.

RESIDENT SECRETARIES AROUSED

Some of our resident secretaries are working tooth and nail to place their respective states in better positions, both in the new-member contest as well as in the membership standing of the states. The best evidence of this that we can present is to be found in the large number of applications being given first listing in the JOURNAL this month. The exact number is 135. Added to the 56 which are given second listing this month, we have a total of 191 new members to be added to the roll between now and the time of the Detroit meeting. Iowa has jumped during the past month from twenty-fourth place to sixth. Virginia has moved up from seventeenth to eighth place. Connecticut has advanced four notches. Idaho has entered the race with six applications and Arkansas and Nevada have done the same with three each. Mississippi shows up this month with five and Rhode Island and South Carolina with one apiece.

Pennsylvania still has eyes on first place, California to the contrary notwithstanding. This month we are listing 42 applications from the Keystone State, against six from the Golden State. Michigan is credited with thirteen.

Dr. John B. Bryant, resident secretary for Iowa, writes as follows:

> Saw your editorial on "Membership Campaign." Iowa will not be missing in the next report, and, if real effort will do it, Iowa will be at the poll when the final reckoning is made this fall.

Dr. I. E. Newsom, resident secretary for Colorado, made the following comment:

> I want to compliment you upon the analysis of the membership in the various states, as I think it will do more than anything else to stimulate activity in this line.

Dr. Edward Records, resident secretary for Nevada, reports as follows:

> All graduate licensed practitioners in the State are members or have applications in.

Dr. W. A. Sullivan, resident secretary for Idaho, reports that all B. A. I. veterinarians in his state are now either members or have applications pending.

DR. GILLILAND DIES

Just as the forms for this issue were being closed, a telegram was received advising us of the death of Dr. S. H. Gilliland, of Marietta, Pa., in St. Joseph's Hospital, Lancaster, Pa., on May 27. A more extended obituary will be published next month.

APPLICATIONS FOR MEMBERSHIP

(See December, 1928, JOURNAL)

FIRST LISTING

ASMUS, R. A. 100 Oakwood Ave., Troy, N. Y.
D. V. M., Cornell University, 1927
Vouchers: J. Elliott Crawford and H. B. Leonard.

ATKINS, HAZEN S. 97 Perry Street, Pontiac, Mich.
D. V. M., Michigan State College, 1923
Vouchers: A. S. Schlingman and Ward Giltner.

AUDINWOOD, HARRY GEORGE Tioga, Pa.
D. V. M., McKillip Veterinary College, 1920
Vouchers: T. E. Munce and John H. Turner.

BAER, LOUIS S. 1099 Elmwood Ave., Columbia, S. C.
D. V. M., Cincinnati Veterinary College, 1918
Vouchers: R. A. Mays and W. K. Lewis.

BARGER, SETH E. 490 N. B. St., Fort Smith, Ark.
D. V. M., Arkansas Veterinary College, 1917
Vouchers: Hugh L. Fry and Joseph H. Bux.

BAVER, ALVIN FRANKLIN 248 E. Main St., Kutztown, Pa.
M. D. C., Chicago Veterinary College, 1904
Vouchers: J. O. Reed and T. E. Munce.

BEATTIE, BENJAMIN MYERS Chambersburg, Pa.
V. M. D., University of Pennsylvania, 1912
Vouchers: Ray O. Whipple and T. E. Munce.

BERRY, HUGH ELMER R. F. D. 6, Staunton, Va.
M. D. V., McKillip Veterinary College, 1911
Vouchers: H. C. Givens and R. E. Brookbank.

BOMAN, THOMAS W. Box 406, Laurel, Miss.
D. V. M., Alabama Polytechnic Institute, 1918
Vouchers: Hartwell Robbins and R. V. Rafnel.

BOYD, HOMER C. Lake Village, Ark.
D. V. M., Kansas State Agricultural College, 1921
Vouchers: Joseph H. Bux and Hugh L. Fry.

BRADLEY, ALFRED MARTIN Alvord, Iowa
D. V. M., Indiana Veterinary College, 1921
Vouchers: Geo. B. Winch and F. E. Schnelle.

BURK, JAMES ARTHUR Shippensburg, Pa.
D. V. M., Ohio State University, 1913
Vouchers: Ray O. Whipple and T. E. Munce.

BURNETTE, CLARENCE ARTHUR 62 Torringford St., Winsted, Conn.
V. S., Ontario Veterinary College, 1916
Vouchers: Geo. E. Corwin and Edwin Laitinen.

BUTTERFIELD, LYNNFORD C. 629 Sinclair St., Reno, Nev.
M. D. C., Chicago Veterinary College, 1902
Vouchers: Edward Records and Lyman R. Vawter.

CALDWELL, CAPT. GEORGE L.
Medical Field Service School, Carlisle Barracks, Pa.
D. V. M., Michigan State College, 1915
Vouchers: R. A. Kelser and R. C. Musser.

CALDWELL, JOHN W. 527 Central Ave., Hemet, Calif.
D. V. M., San Francisco Veterinary College, 1915
Vouchers: W. L. Curtis and J. P. Bushong.

CAMPBELL, LARKIN S. 1629 Fillmore, Topeka, Kans.
D. V. S., Kansas City Veterinary College, 1904
Vouchers: N. L. Townsend and C. B. Clement.

CARD, BOWEN OLIVER — Sylvania, Pa.
V. S., Ontario Veterinary College, 1913
Vouchers: John H. Turner and T. E. Munce.

CARR, FRANK H. — 722 State Office Bldg., Lansing, Mich.
D. V. M., Indiana Veterinary College, 1921
Vouchers: M. P. Hunt and T. S. Rich.

CHURCH, DWIGHT S. — 626 Liberty St., Erie, Pa.
V. S., Ontario Veterinary College, 1895
Vouchers: J. B. Reidy and T. E. Munce.

COOVER, EUGENE M. — 147 West 18th St., Erie, Pa.
M. D. C., Chicago Veterinary College, 1911
Vouchers: M. P. Hendrick and E. E. Bittles.

CORKILL, JOSEPH J. — 317 Patterson St., Lansford, Pa.
D. V. S., Grand Rapids Veterinary College, 1907
Vouchers: J. O. Reed and T. E. Munce.

DAVIES, MONTAGUE A. — Troy, Pa.
V. S., Ontario Veterinary College, 1890
Vouchers: John H. Turner and T. E. Munce.

DAVIS, CHARLES LOUIS — 303 Livestock Exchange Bldg., Denver, Col.
D. V. M., Colorado Agricultural College, 1921
Vouchers: T. A. Shipley and Robert B. Leeper.

DENHAM, WILLIAM BURTON — Cumberland, Iowa
D. V. M., Kansas City Veterinary College, 1914
Vouchers: F. D. Weimer, C. E. Harry and John B. Bryant.

DENSON, LEONIDAS L. JR. — Box 42, Ackerman, Miss.
D. V. M., Alabama Polytechnic Institute, 1922
Vouchers: Hartwell Hobbins and Edgar Heiny.

DERK, MILES RAYMOND — 124 S. Broad St., Jersey Shore, Pa.
V. M. D., University of Pennsylvania, 1913
Vouchers: John H. Turner and T. E. Munce.

DE TURCK, DANIEL H. — Oley, Pa.
M. D. C., Chicago Veterinary College, 1907
Vouchers: J. O. Reed and T. E. Munce.

DICKMAN, ANDREW JACKSON — 1120 N. 6th Ave., Boise, Idaho
D. V. M., Colorado Agricultural College, 1915
Vouchers: W. A. Sullivan and J. E. Ellis.

DEFENDORF, HORATIO ERNEST — Grand Blanc, Mich.
M. D. C., Chicago Veterinary College, 1909
Vouchers: A. S. Schlingman and B. J. Killham.

DOCKSTADER, HALL ANDREW — Rudd, Iowa
D. V. M., Indiana Veterinary College, 1922
Vouchers: Chris. E. Juhl and Grant B. Munger.

DONOVAN LT. COL. ANDREW E.
c/o Adjutant General, U. S. Army, Washington, D. C.
D. V. S., San Francisco Veterinary College, 1902
Vouchers: R. A. Kelser and H. J. Juzek.

DRAKE FRANCIS B. — 3630 Edwards Road, Hyde Park, Cincinnati, Ohio
D. V. M Cincinnati Veterinary College, 1904
Vouchers: W. W. Renter and John W. Hermann.

DUNN, RALPH ALEXANDER — 511 East Water St., Charlottesville, Va.
D. V. S., American Veterinary College, 1891
Vouchers: H. C. Givens and R. E. Brookbank.

DYKEMA, HERMAN — R. No. 4, Muskegon, Mich.
D. V. M., Michigan State College, 1928
Vouchers: Edw. K. Sales and B. J. Killham.

EHLERS, DANIEL P. — Box 403, Harrisburg, Pa.
D. V. M., Kansas State Agricultural College, 1928
Vouchers: A. L. Brueckner and M. F. Barnes.

ELIASON, THEODORE O. Kerkhoven, Minn.
D. V. S., Western Veterinary College, 1906
Vouchers: H. C. H. Kernkamp and R. Fenstermacher.

EMERY, ASHTON W. 97 Perry St., Pontiac, Mich.
D. V. M., Michigan State College, 1923
Vouchers: A. S. Schlingman and Ward Giltner.

FENSTERMACHER, JAMES O. New Tripoli, Pa.
M. D. V., McKillip Veterinary College, 1906
Vouchers: J. O. Reed and T. E. Munce.

FISHER, WILLIAM F. Box 155, Yerington, Nev.
D. V. M., Colorado Agricultural College, 1923
Vouchers: Edward Records and Lyman R. Vawter.

FITZ GERALD, GERALD W.
Army Vet. School, Army Med. Center, Washington, D. C.
D. V. M., Kansas State Agricultural College, 1916
Vouchers: R. R. Dykstra and R. A. Kelser.

FORD, JOHN ARNOLD 909 W. Anaheim St., Long Beach, Calif.
V. S., Ontario Veterinary College, 1910
Vouchers: W. L. Curtis and C. C. Oderkirk.

FOSS, OLE D. Christine, N. Dak.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: Lee M. Roderick and A. F. Schalk.

GAGE, FRED B. 1822 N. Alexandria Ave., Los Angeles, Calif.
M. D. V., Harvard University, 1896
Vouchers: W. L. Curtis and J. P. Bushong.

GARRAHAN, LORENZO P. Florida 668, Buenos Aires, Argentina.
D. V. M., Ohio State University, 1908
Vouchers: Adolph Eichhorn and David S. White.

GAW, JOHN HOWARD Whytheville, Va.
D. V. M., Ontario Veterinary College, 1907
Vouchers: H. C. Givens and R. E. Brookbank.

GENRE, T. T. 722 State Office Bldg., Lansing, Mich.
D. V. M., Indiana Veterinary College, 1921
Vouchers: M. P. Hunt and T. S. Rich.

GIFFEE, JOE W. Starkville, Miss.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: R. A. Ramsay and Hartwell Robbins.

GILBERT, I. P. Courtland, Va.
D. V. M., U. S. College of Veterinary Surgeons, 1922
Vouchers: H. C. Givens and R. E. Brookbank.

GILDOW, E. M. University of Idaho, Moscow, Idaho
D. V. M., State College of Washington, 1922
Vouchers: W. A. Sullivan and W. E. Neary.

GREER, LT. CHARLES S. Fort Bliss, El Paso, Texas
D. V. M., Iowa State College, 1926
Vouchers: R. A. Kelser and R. C. Musser.

HAINES, W. ALBERTSON Bristol, Pa.
V. M. D., University of Pennsylvania, 1907
Vouchers: Henry W. Turner and T. E. Munce.

HANNON, JOSEPH 130 W. 6th St., Davenport, Iowa
M. D. V., McKillip Veterinary College, 1910
Vouchers: J. A. Barger and B. L. Dawson.

HARRIS, FRANK THOMAS Box 852, Twin Falls, Idaho
D. V. M., State College of Washington, 1928
Vouchers: W. A. Sullivan and H. R. Groome.

HENDERSON, CLARENCE B.
c/o Leavenworth Pkg. & Stor. Co., Leavenworth, Kans.
D. V. S., Kansas City Veterinary College, 1909
Vouchers: F. S. Bingham and Chas. F. Pinkham.

HERROLD, WILLIAM C. 3129 Spring Grove Ave., Cincinnati, Ohio
V. S., Ohio State University, 1907
Vouchers: W. W. Renter and John W. Hermann.

HIATT, WILLIAM L.
212 Live Stock Exch. Bldg., Stock Yds. Sta., Oklahoma City, Okla.
M. D. C., Chicago Veterinary College, 1902
Vouchers: A. H. Denham and L. J. Allen.

HOBDY, WILLIAM MADISON Box 15, Salem, Va.
D. V. M., Alabama Polytechnic Institute, 1911
Vouchers: H. C. Givens and R. E. Brookbank.

HOFFMIRE, COLENZO H. Franklin Alley, Adrian, Mich.
D. V. M., Ohio State University, 1908
Vouchers: A. S. Schlingman and B. J. Killham.

HORNER, ERNEST ALLEN Brighton, Iowa
D. V. M., McKillip Veterinary College, 1919
Vouchers: Henry Hell and John B. Bryant.

HOVORKA, JOSEPH W. 6213 W. 22nd St., Berwyn, Ill.
D. V. M. McKillip Veterinary College, 1919
Vouchers: Herman Busman and Albert J. Noonan.

HURD, RAY B. Box 202, Nampa, Idaho
D. V. S., Kansas City Veterinary College, 1908
Vouchers: W. A. Sullivan and A. T. Kinsley.

JOHNSON, E. E. 4529 Lodewyck, Detroit, Mich.
D. V. M., Michigan State College, 1926
Vouchers: Hugo Cornehl and John Hoberg.

KEELOR, ALLEN Z. Gratersford, Pa.
V. S., Ontario Veterinary College, 1891
Vouchers: C. O. Neuhaus and T. E. Munce.

KEMP, DONALD T. Dept. of Health, 1300 Beaubien St., Detroit, Mich.
B. V. S. Ontario Veterinary College, 1910
Vouchers: Hugo Cornehl and A. S. Schlingman.

KINGAN, JOHN C. 4082 Liberty Ave., Pittsburgh, Pa.
V. S., Ontario Veterinary College, 1896
Vouchers: Wm. Brod and T. E. Munce.

KLUG, JOHN HENRY Randolph Lake, Wis.
D. V. M., Grand Rapids Veterinary College, 1917
Vouchers: James S. Healy and H. D. Lazelere.

LAWTON, RUSSELL G. 310 State House, Providence, R. I.
V. S., New York College of Veterinary Surgeons, 1895
Vouchers: Joseph S. Barber and Thos. E. Robinson.

LEWIS, CAPT. RALPH HENRY Fort Sam Houston, Texas
D. V. M., George Washington University, 1917
Vouchers: R. A. Kelser and R. C. Musser.

LITTLE, HARRY JOSEPH 515 Market St., Williamsport, Pa.
V. M. D., University of Pennsylvania, 1912
Vouchers: John H. Turner and R. G. Little.

LOSSIE, FRED H. Harbor Creek, Pa.
M. D. C., Chicago Veterinary College, 1912
Vouchers: M. P. Hendrick and E. E. Bittles.

LUBBEHUSEN, RAYMOND ELMER Box 403, Harrisburg, Pa.
B. S., North Dakota Agricultural College
D. V. M., Ohio State University, 1918
Vouchers: M. F. Barnes and T. E. Munce.

LUDINS, GEORGE H. 203 Affleck St., Hartford, Conn.
D. V. M., Cornell University, 1924
Vouchers: Edwin Laitinen and Wallace F. Vail.

LYNCH, WALTER P. Towanda, Pa.
V. M. D., University of Pennsylvania, 1918
Vouchers: T. E. Munce and John H. Turner.

MCCABE, JAMES C. Box 211, Iowa City, Iowa
D. V. M., Chicago Veterinary College, 1913
Vouchers: Grant B. Munger and John B. Bryant.

MCCOY, ELLIS E. 408 Park St., Charlottesville, Va
D. V. S., Kansas City Veterinary College, 1907
Vouchers: H. C. Givens and R. E. Brookbank.

MADSEN, DAVID E. Box 403, Harrisburg, Pa.
D. V. M., Colorado Agricultural College, 1928
Vouchers: M. F. Barnes and T. E. Munce.

MICHENER, HIRAM M. North Wales, Pa.
V. M. D., University of Pennsylvania, 1912
Vouchers: T. E. Munce and C. O. Neuhaus.

MILLER, FRANK G. 719 8th Ave., Lewiston, Idaho
D. V. M., Iowa State College, 1908
Vouchers: W. A. Sullivan and A. J. Powell.

MITCHELL, GEORGE ELMER 3330 Eastside Ave., Cincinnati, Ohio
D. V. M., Cincinnati Veterinary College, 1915
Vouchers: Norton Dock and John W. Hermann.

MUSSELMAN, EUGENE E. King and Queen Court House, Va.
D. V. M., U. S. College of Veterinary Surgeons, 1926
Vouchers: H. C. Givens and R. E. Brookbank.

NISSLEY, S. M. 16 Spring St., Bellefonte, Pa.
V. M. D., University of Pennsylvania, 1904
Vouchers: Ira Mitterling and T. E. Munce.

OWENS, LT. JOHN LLOYD Fort Sam Houston, Texas
D. V. M., Colorado Agricultural College, 1926
Vouchers: R. C. Musser and R. A. Kelser.

PEASE, W. W. 448 North St., Meadville, Pa.
M. D. C., Chicago Veterinary College, 1911
Vouchers: M. P. Hendrick and E. E. Bittles.

PILGRIM, RALPH V. Clarksville, Texas
D. V. M., Kansas City Veterinary College, 1915
Vouchers: H. L. Darby and Jesse L. Shabram.

PLESS, LOUIS R. 16630 Washburn Ave., Detroit, Mich.
D. V. M., Michigan State College, 1920
Vouchers: Hugo Cornehl and John Hoberg.

POSEINER, WM. 922 Nassau St., Cincinnati, Ohio
D. V. M., Cincinnati Veterinary College, 1913
Vouchers: Norton Dock and John W. Hermann.

POWELL, THOMAS MAURICE 27 E. Markison Ave., Columbus, Ohio
D. V. M., Indiana Veterinary College, 1924
Vouchers: F. A. Lambert and R. N. Birdwhistell.

RAMSEY, CAPT. MOTT. Fort Ringgold, Texas
D. V. M., McKillip Veterinary College, 1916
Vouchers: R. A. Kelser and R. C. Musser.

RASMUSSEN, GEORGE C. Kimballton, Iowa
M. D. C., Chicago Veterinary College, 1907
Vouchers: C. J. Graf, C. E. Harry and John B. Bryant.

RATTS, FLOYD S. 1024 Laramie, Manhattan, Kans.
D. V. M., Kansas State Agricultural College, 1922
Vouchers: F. A. Imler and C. B. Clement.

REA, HERBERT E. West Branch, Mich.
V. S., Ontario Veterinary College, 1902
Vouchers: A. S. Schlingman and E. E. Patterson.

READING, CLAUDE HARVEY Hopkins, Mich.
D. V. M., Michigan State College, 1927
Vouchers: A. S. Schlingman and Ward Giltner.

REAGOR, HARRY ABNER 30 California Ave. Reno, Nev.
D. V. S., Kansas City Veterinary College, 1906
Vouchers: Lyman R. Vawter and Edward Records.

REDDERT, FRED E. Independence, Calif.
D. V. M., Colorado Agricultural College, 1928
Vouchers: W. L. Curtis and J. P. Bushong.

REED, CHARLES W. R. F. D. 4, Gibsonia, Pa.
V. M. D., University of Pennsylvania, 1913
Vouchers: Wm. Brod and T. E. Munce.

REICHLEY, JOHN RALPH Dover, Pa.
V. S., Ontario Veterinary College, 1915
Vouchers: Ray O. Whipple and T. E. Munce.

RICHEY, ALFRED N. Lanesboro, Iowa
D. V. M., Iowa State College, 1924
Vouchers: C. H. Stange and John B. Bryant.

RILE, EDWARD A. Blue Bell, Pa.
V. M. D., University of Pennsylvania, 1906
Vouchers: T. E. Munce and C. O. Neuhaus.

RITTER, A. B. Pennsburg, Pa.
M. D. C., Chicago Veterinary College, 1907
Vouchers: J. B. Reidy and C. O. Neuhaus.

RIVERS, REUBEN N. Lansdale, Pa.
V. M. D., University of Pennsylvania, 1901
Vouchers: C. O. Neuhaus and T. E. Munce.

ROSEKRANS, WILLIAM R. 1210 Evergreen Ave., Bronx, New York, N. Y.
D. V. S., American Veterinary College, 1896
Vouchers: D. R. Gillies and James Fleming.

ROUSE, PALMER LADD 2203 Peach St., Erie, Pa.
B. V. Sc., Ontario Veterinary College, 1923
Vouchers: M. P. Hendrick and E. E. Bittles.

RUBY, JOHN LOUIS 4036 Platt Ave., Fresno, Calif.
D. V. M., San Francisco Veterinary College, 1916
Vouchers: John McInnes and Joseph M. Arburua.

RUDOLPH, JAMES ABRAM 341 Terrace Ave., Cincinnati, Ohio
D. V. M., Cincinnati Veterinary College, 1911
Vouchers: Norton Dock and John W. Hermann.

RUTH, VINCENT MOYER Franconia, Pa.
V. S., Ontario Veterinary College, 1913
Vouchers: T. E. Munce and C. O. Neuhaus.

SHERRICK, GEORGE D. Smithfield Hotel, Smithfield, Pa.
V. M. D., University of Pennsylvania, 1918
Vouchers: J. B. Reidy and T. E. Munce.

SHIMER, W. S. Box 355, Altoona, Pa.
V. S., Ontario Veterinary College, 1885
Vouchers: Ira Mitterling and T. E. Munce.

SKELTON, JAMES A. Prairie Grove, Washington Co., Ark.
D. V. M., St. Joseph Veterinary College, 1914
Vouchers: Joseph H. Bux and Hugh L. Fry.

SMITH, HILTON A. State College of Washington, Pullman, Wash.
M. D. V., Colorado Agricultural College, 1928
Vouchers: E. E. Wegner and N. G. Covington.

SMITH, WILBUR C. 219 East 10th St., Waterloo, Iowa
D. V. M., Iowa State College, 1909
Vouchers: J. E. Shelton and L. T. Langland.

SNYDER, JOHN C. Elderton, Pa.
D. V. M., Cincinnati Veterinary College, 1914
Vouchers: T. A. Marshall and T. E. Munce.

STEIN, CLARENCE DINSMORE Box 586, Pearl River, N. Y.
V. M. D., University of Pennsylvania, 1911
Vouchers: B. M. Lyon and Frederick A. Grenfell.

STILL, SEABORN HASSELL Box 393, Suffolk, Va.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: Wm. Moore, W. C. Dendinger and H. C. Givens.

STRICKLER, JACOB ETTER Chambersburg, Pa.
D. V. S., U. S. College of Veterinary Surgeons, 1911
Vouchers: Ray O. Whipple and T. E. Munce.

STURROCK, ALEXANDER P. Waterford, Pa.
D. V. M., Cornell University, 1917
Vouchers: M. P. Hendrick and Earle S. Pickup.

SUPER, DANIEL H. Warrensville, Pa.
V. S., Ontario Veterinary College, 1895
Vouchers: John H. Turner and T. E. Munce.

TAYLOR, BRAINARD LOUIS Emmett, Idaho
D. V. M., Kansas State Agriculture College, 1920
Vouchers: W. A. Sullivan and W. E. Neary.

TELFORD, ROSWELL A. 914 Reber St., Waterloo, Iowa
D. V. M., St. Joseph Veterinary College, 1917
Vouchers: J. E. Shelton and L. T. Langland.

TIERNEY, DANIEL DAVID 8055 S. Bishop St., Chicago, Ill.
M. D. V., McKillip Veterinary College, 1908
Vouchers: Mihalz Borsos and Albert J. Noonan.

UNDERWOOD, PAUL CLIFFORD 4409 Sidney Ave., Chicago, Ill.
V. S., B. V. Sc., Ontario Veterinary College, 1926
Vouchers: J. V. Lacroix and H. Busman.

UNERTL, JOHN T. 380 4th St., Milwaukee, Wis.
D. V. S., Chicago Veterinary College, 1892
Vouchers: James S. Healy and H. D. Larzelere.

UPTON, CLARENCE c/o Evansville Packing Co., Evansville, Ind.
D. V. M., Kansas City Veterinary College, 1915
Vouchers: H. J. Hoyman and W. H. Gruner.

VAN VLANDREN, CORNELIUS 610 Rubber Ave., Naugatuck, Conn.
V. S., Ontario Veterinary College, 1903
Vouchers: Edwin Laitinen and Thomas Bland.

VANDERLOO, VIVIAN BERNARD 870 West 14th St., Dubuque, Iowa.
D. V. M., Iowa State College, 1921
Vouchers: H. J. Hagerty and Charles J. O'Brien.

WADDLE, HARRY PATTON 401 Walnut St., Hattiesburg, Miss.
D. V. M., Kansas City Veterinary College, 1910
Vouchers: R. A. Ramsay and Hartwell Robbins.

WAGNER, C. ARTHUR 1210 Park Ave., Williamsport, Pa.
V. M. D., University of Pennsylvania, 1914
Vouchers: J. B. Reidy and B. Scott Tritz.

WALTER, HARRY K. 318 Livestock Exch., Herrs Island, Pittsburgh, Pa.
V. M. D., University of Pennsylvania, 1893
Vouchers: A. E. Wight, Elmer Lash and J. B. Reidy.

WARWICK, JOHN G. 3442 S. 15th Ave., Seattle, Wash.
D. V. M., Grand Rapids Veterinary College, 1911
Vouchers: D. S. Kay and C. L. Norris.

WATERS, CAPT. FRED C. Presdio of Monterey, Calif.
D. V. M., Ohio State University, 1912
Vouchers: R. A. Kelser and H. J. Juzek.

WEBB, EDW. A. 513 N. Pennsylvania Ave., Avondale, Pa.
V. M. D., University of Pennsylvania, 1927
Vouchers: E. L. Stubbs and Benjamin Price.

WHYTE, WILLIAM BURNETTE 9 Gerald Ave., Highland Park, Mich.
B. V. Sc., Ontario Veterinary College, 1924
Vouchers: A. S. Schlingman and H. Preston Hoskins.

WIGHT, CAPT. ALLEN C. Fort Sam Houston, Texas
D. V. M., Ohio State University, 1912
Vouchers: Lawrence R. Bower and Ray J. Stanclift.

WOODS, JOHN W. Mendenhall, Miss.
D. V. M., Kansas City Veterinary College, 1914
Vouchers: Hartwell Robbins and R. V. Rafnel.

YOHNG, JOHN O. 626 Clay St., Topeka, Kans.
D. V. S., Kansas City Veterinary College, 1895
Vouchers: F. A. Imler and C. B. Clement.

Applications Pending

SECOND LISTING

Blamey, Edwin Reginald, 17 West 71st St., New York City, N. Y.
Bottorff, Charles A., Jr., University of New Hampshire, Durham, N. H.
Boyd, Robert, Box 53, Mill Valley, Calif.
Brandner, Walter E., Petaluma, Calif.
Breininger. Chas. B., 624 Goldsborough St., Easton, Md.
Britten, Mart S., Box 327 Harrisburg, Pa.
Bushong, Jesse W., West Grove, Pa.
Clarkson, P. V., Box 327, Harrisburg, Pa.
Colflesh, Joseph Harmon, 537 Hugart St., Confluence, Pa.
Cotton, Harry L., 18479 Trinity Ave., Detroit, Mich.
Craven, Harry T., Reynoldsville, Pa.
Crawford, James Stuart, 708 Broadway, Far Rockaway, L. I., N. Y.
Crawford, John Elliott, 708 Beach 19th St., Far Rockaway, L. I., N. Y.
Darling, Robert, 3630 Georgia St., San Diego, Calif.
Dick, Thomas Crawford, Box 104, Mineola, N. Y.
Dodge, Loren N., 219 N. Massey St., Watertown, N. Y.
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Elkow, Stephen R., 13153 Hamilton Ave., Highland Park, Mich.
Engle, Joseph B., Box 432, Summit, N. J.
Feelyater, Earll Milton, Randolph, Wis.
Graham, Donald, Box 198, Gonzales, Calif.
Grinnells, C. D., State College Station, Raleigh, N. C.
Gurney, Harry L., 3994 Iowa St., San Diego, Calif.
Haney, William Francis, 1414—9th St., Modesto, Calif.
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Jensen, Thomas Jelmer, Arkdale, Wis.
Johnson, Joseph, Union Stock Yards, Lancaster, Pa.
Kutz, Harry Cooper, Mansfield, Pa.
Lynch, Raymond J., Dushore, Pa.
McGroarty, William V., Smethport, Pa.
McIntyre, Howard Hoke, Box 1013, Detroit, Mich.
Milo, Howard Alfred, 458 Ella St., Williamsburg, Pa.
Morris, Eralsey Charles, 3650 F St., Eureka, Calif.
Moulton, Cecil, 1494 4th St., San Rafael, Calif.
Murdoch, James W., Capitol Station, Helena, Montana
Pease, Ray Herbert, Box 553, Los Banos, Calif.
Pfeiffer, Walter F., R. 5., Plymouth, Wis.
Pickering, Clifford E., Fort Mason, Calif.
Pier, B. Clark, El Heusah Apts., Dover, Del.
Plank, Neil, 332 Federal Bldg., Lincoln, Nebr.

Poley, Philip P., 33 Abeel St., Kingston, N. Y.
Quarll, Joseph T., R. D. No. 1, Avondale, Pa.
Ramler, John Verlin, Box 85, Wheaton, Minn.
Robenstein, C. C., Hugo, Colo.
Ruddle, Olin Harrison, Box 23, Salem, Va.
Schuey, George Bernard, 526 Todd St., Wilkinsburg Sta., Pittsburgh, Pa.
Smith, Stanley N., 501 Rollins St., Columbia, Mo.
Stader, Otto, 2318 E. Johnson, Madison, Wis.
Taylor, H. Hawyard, 101 W. 2nd St., Media, Pa.
Thomas, John Joseph, 101 Welles St., Forty Fort, Pa.
Thompson, Arvo Theodore, Box 403, Harrisburg, Pa.
Urich, John Russell, Rossville, Pa.
Wiley, J. Ross, 705 North Shippen St., Lancaster, Pa.
Worrell, Temp Jefferson, Box 1725, Pampa, Tex.
Yoder, Edwin C., Birdsboro, Pa.
Zimmerman, Elijah Robert, Mifflinburg, Pa.

The amount which shall accompany an application this month is $7.91 which covers membership fee and dues to January 1, 1930, including subscription to the JOURNAL.

Drive to Dynamic Detroit.

COMING VETERINARY MEETINGS

California State Veterinary Medical Association. Sequoia National Park, Calif. June 10-12, 1929. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Eastern States Tuberculosis Eradication Conference. Municipal Auditorium, Springfield, Mass. June 18-19, 1929. Dr. E. A. Crossman, Chairman, Program Committee, 2001 Customhouse Bldg., Boston, Mass.

Massachusetts Veterinary Association. Eastern State Exposition Grounds, Springfield, Mass. June 20, 1929. Dr. Harry W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Texas, State Veterinary Medical Association of, and Texas A. & M. College Short Course for Veterinarians. A. & M. College of Texas. June 17-20, 1929. Dr. D. Pearce, Secretary, Leonard, Texas.

New York State Veterinary Medical Society. Ithaca, N. Y. June 19-20, 1929. Dr. C. E. Hayden, Secretary, 110 Irving Place, Ithaca, N. Y.

Michigan State Veterinary Medical Association. East Lansing, Mich. June 25, 1929. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing, Mich.

Missouri Veterinary Medical Association. State Fair Grounds, Sedalia, Mo. June 25-27, 1929. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

North Carolina State Veterinary Medical Association. Asheville, N. C. June 26-27-28, 1929. Dr. W. T. Scarborough, Secretary, 320 S. Blount St., Raleigh, N. C.

Manitoba, Veterinary Medical Association of. Brandon, Man. July 5, 1929. Dr. Wm. Hilton, Secretary, 510 Greenwood Place, Winnipeg, Man.

Montana Veterinary Medical Association. Missoula, Mont. July 8-9, 1929. Dr. Hadleigh Marsh, Secretary, Helena, Mont.

Missouri Valley Veterinary Association. Omaha, Nebr. July 8-10, 1929. Dr. E. R. Steel, Secretary, 8023 Wornall Rd., Kansas City, Mo.

Kentucky Veterinary Medical Association. Louisville, Ky. July 10-11, 1929. Dr. C. G. Kriedler, Secretary, Maysville, Ky.

Minnesota State Veterinary Medical Association. University Farm, St. Paul, Minn. July 10-11, 1929. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.

North Dakota Veterinary Medical Association. Fargo, N. Dak. July 11-12, 1929. Dr. Lee M. Roderick, Secretary, State College Station, Fargo, N. Dak.

New Jersey, Veterinary Medical Association of. Asbury Park, N. J. July 11-12, 1929. Dr. E. R. Cushing, Secretary, Box 536, New Brunswick, N. J.

Wisconsin Veterinary Medical Association. Wisconsin Rapids, Wis. July 15-16, 1929. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

British Columbia, Washington and Oregon Veterinary Medical Associations. Vancouver, B. C. July 15-17, 1929. Dr. Clifford Ackley, Secretary, Winlock, Wash.

Nevada State Veterinary Association. Reno, Nevada. July 17, 1929. Dr. Edward Records, Secretary, University of Nevada, Reno, Nevada.

South Carolina Association of Veterinarians. Hartsville, S. C. July 16-17, 1929. Dr. M. R. Blackstock, Secretary, 157 Hampton Ave., Spartansburg, S. C.

Maryland State Veterinary Medical Association. Medical Hall, 1211 Cathedral St., Baltimore, Md. July 19-20, 1929. Dr. E. M. Pickens, Secretary, College Park, Md.

Illinois State Veterinary Medical Association. Jefferson Hotel, Peoria, Ill. July 17-18, 1929. Dr. W. H. Welch, Secretary, Lexington, Ill.

Virginia State Veterinary Medical Association. Hotel George Washington, Winchester, Va. July 25-26, 1929. Dr. George C. Faville, Secretary, Hampton, Va.

JOHNIN VERSUS AVIAN TUBERCULIN AS A DIAGNOSTIC AGENT FOR PARATUBERCULOSIS (JOHNE'S DISEASE) OF CATTLE

By W. A. HAGAN *and* A. ZEISSIG

Department of Pathology and Bacteriology, New York State Veterinary College, Cornell University, Ithaca, N. Y.

In 1895, Professor Johne,[8] of the Dresden Veterinary High School, discovered that a form of chronic dysentery which he observed in a cow was due to an acid-fast organism. He supposed he was dealing with a form of tuberculosis but when guinea pigs which were inoculated with the bacillus-containing material failed to contract the disease, he was forced to conclude that the organism could not be an ordinary tubercle organism. He thought that it might be an attenuated bovine type tubercle bacillus or an avian type. He inclined to the latter belief but the matter was not definitely settled.

In the first decade of the twentieth century, many workers found that the disease which had been described by Johne, in collaboration with Frothingham, was wide-spread. In 1906, it was proposed by Bang[1] that it be designated *paratuberculosis*. A little later M'Fadyean[9] proposed the name "Johne's disease," feeling that the term paratuberculosis was misleading, since the disease had nothing to do with tuberculosis.

All attempts to cultivate the causative organism were fruitless until 1911. Before this time many inoculation experiments were carried out, usually with the purpose of finding whether it was, or was not, a form of the tubercle organism. Most of these failed completely but some resulted in the inoculated animals developing tuberculosis, probably because the material for inoculation came from animals which were affected with tuberculosis as well as the dysentery. In 1906, however, B. Bang[1] succeeded in producing typical cases of the disease in young calves by feeding infected material from the intestines of diseased animals. As a result of his experiences with the disease, Bang inclined strongly to the belief that the disease had nothing to do with tuberculosis but was a separate and distinct entity. Many others reached this conclusion also but there were some who held to the belief that the disease was a peculiar form of tuberculosis. This idea was

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

finally completely dispelled when Twort,[15] in 1911, announced his success in cultivating the organism artificially. Using his technic, others were successful and at the present time it can fairly be accepted that the organism is not identical with any of the tubercle bacillus types and the disease has nothing to do with tuberculosis.

Johne's disease, as the affection is generally known in English-speaking countries, is recognized as a wide-spread, incurable disease of cattle. It is very destructive in most of the countries of western Europe and has recently been reported by Edwards[6] as a most serious disease of cattle in India. In the United States the disease has been reported in at least 33 states, but it is probable that it is not at all prevalent in more than half a dozen of these. It is known to be rather prevalent in Pennsylvania, New Jersey, Maryland, New York and Wisconsin. It is probably more prevalent in other states than is commonly thought, since it is not recognized in many instances.

The causative organism of Johne's disease is discharged with the feces. It is not known whether all infected animals discharge the organism constantly or not. It is probable that they do not. Sometimes enormous numbers of the bacillus are discharged with the bowel content. In other cases, even though the animal may be actively scouring, the organisms are found with difficulty or not at all.

The disease does not appear to spread as rapidly as might be supposed. Even though a herd may have harbored the infection for years, it is not often that more than 10 per cent of the animals will be lost from it in any one year. It is now known, however, that in such herds a considerably greater percentage will usually react to allergic tests and at autopsy show evidence of the infection. It is fairly certain that many of these animals, perhaps all of them, will have been eliminating the infection for months or years before they show any clinical evidence of the disease. In order that the disease may be brought under control it is thus necessary that means be developed to detect the infection in animals before they are sufficiently advanced in the disease to show clinical evidence of it.

Before the identity of the causative agent was known, it was discovered that ordinary tuberculin was of no value for this purpose. Thus B. Bang[2] reported that in many herds in Denmark there was no trace of tuberculosis and no reactors to the tuberculin test, yet Johne's disease ravaged them. O. Bang[3] reported

that doses of ordinary tuberculin, in amounts as great as 20 cc, had failed to produce reactions in animals affected with Johne's disease.

In 1919, O. Bang[3] attempted to prepare a substance analogous to tuberculin by extracting with glycerin an active principle from scrapings of intestinal mucous membranes which were very rich in acid-fast bacilli. Some success was met with, but the method did not appear to be generally useful and was quickly abandoned. He next tested a tuberculin made from the avian type of tubercle bacillus and discovered that this substance gave temperature reactions in many cattle which showed symptoms of Johne's disease and in others which had not yet shown symptoms but which later proved to be infected. The product was used subcutaneously. The temperature rise was usually not quite so great as that obtained by the use of tuberculin in tuberculous animals.

When he had succeeded in cultivating the causative organism of the disease, Twort immediately set about the preparation of a specific product analogous to tuberculin. In this he met with considerable difficulty, inasmuch as the Johne bacillus failed to develop a luxuriant growth in any type of fluid medium. He nevertheless prepared and tested some preparations, and found, as might be expected, that they would cause temperature reactions when injected subcutaneously. Later M'Fadyean, Sheather and Edwards[10,11] reported the results of their experiments with "johnin," as they christened the product. Still later, Beach and Hastings[4] in this country reported the results of their work with johnin. In all of these instances, however, the workers labored under the difficulty that they could not obtain uniformly luxuriant growths of the causative organism and as a result their work, for the most part at least, may be said to have been carried out with rather weak products.

Almost simultaneously, in 1928, there appeared papers by Wright[17] and by Boquet[5] independently describing new and far more successful methods of cultivating Johne's organism than had been available previously. Hastings, Beach and Mansfield[7] also have been much more successful of late in obtaining heavy growths of the organism on fluid media. In all of these instances synthetic media have been substituted for the beef infusion broth which formed the basis of the older media. The johnin prepared by the use of newer media should be of far greater potency than

any of the older products and the reports which have appeared so far regarding them are favorable (Wright,[17] Turner[14]).

In the course of our own work on Johne's disease, we encountered the same difficulty that had beset all others before us, namely, the difficulty in preparing a potent johnin. Rather peculiarly, cultures of this organism could be maintained without great difficulty on solid media but on a fluid medium growth was always exceedingly erratic and usually scanty. A fairly useful johnin was prepared by extracting the solid cultures with distilled water in the refrigerator but this is laborious and inexact. We accordingly turned our thoughts to the properties of avian tuberculin, and the efficiency of this substance as a diagnostic agent in the disease.

Since the successful isolation of Johne's bacillus, all workers have centered their attention upon the use of a diagnostic product prepared from this organism, and avian tuberculin has received scant attention. This, of course, is natural, since there is little in common between avian tuberculosis and Johne's disease and one would assume that there would be little in common between their respective organisms. It seems to have been quite generally assumed that, although there was no question about the fact that avian tuberculin would sometimes give reactions in animals affected with Johne's disease, such a non-specific product could hardly be expected to be as reliable as one made from the specific organism. That this reasoning was logical we are not prepared to deny, and yet we believe that the biochemic relationship between the avian tubercle organism and the organism of Johne's disease is closer than has hitherto been suspected, and that tuberculin prepared from the avian type tubercle bacillus is more reliable as a diagnostic agent for Johne's disease than has previously been thought.

That animals affected with Johne's disease will react fairly regularly to doses of avian tuberculin and will not to ordinary tuberculin or to "tuberculins" prepared from other acid-fast organisms is a remarkable fact. That group reactions occur among organisms of similar nature is, of course, a well-known fact. Thus we have agglutinins which will react with organisms other than the species which stimulated their production, and it appears that almost any acid-fast organism will fix complement in the presence of tuberculous sera. But the relationship between the two organisms in question is not a group reaction in the ordinary

sense of the term, inasmuch as it is not participated in by other acid-fast organisms as far as is known at present.

We have used avian tuberculin as well as johnin on herds naturally infected with Johne's disease, and have conducted a number of experiments in which animals were sensitized with either the avian tubercle organism or Johne's bacillus and subsequently tested with both avian tuberculin and johnin. A report of some of this work will be given.

Reciprocal Sensitization of Guinea Pigs to Avian Tuberculin and Johnin by Injection of the Avian Tubercle Organism and the Johne Organism

Inoculation of guinea pigs with pure cultures or with tissues rich in the organism of Johne's disease generally results in the formation of an abscess if the injection be subcutaneous, or of small nodules on the peritoneum if the injection be intraperitoneal. The animals are never appreciably affected by the treatment. The avian tubercle organism behaves similarly but acts more severely. Usually the animals emaciate and some may die. At autopsy of these animals, tubercles are not usually found but often organisms may be found in smears from some of the internal organs.

Ten guinea pigs were selected from the stock. Their abdomens were shaved and intradermal injections of concentrated johnin and avian tuberculin were made at separate points. The johnin was of our own manufacture (lot XIV), a product prepared from cultures on solid media. The avian tuberculin was produced in the usual manner. At the end of 48 hours the animals were inspected. The johnin appeared to have almost no non-specific action on the skin, while the tuberculin had very little.

Five of the animals were now injected in the right thigh with a heavy suspension of the Johne bacillus in sterile mineral oil. The organisms had been washed from the surface of an agar culture. The other five animals were treated similarly with a suspension of the avian tubercle bacillus which had been prepared in a similar manner. These animals were subsequently given three intradermal tests, using the same tuberculin and johnin as before. The results of these tests are given in table I.

It will be noted that the animals inoculated with the avian type tubercle organism quickly became sensitized, while those receiving the Johne bacillus were rather slow to react. The dose of the two organisms was approximately the same, hence it seems

TABLE I—*Results of simultaneous intradermal tests with johnin and avian tuberculin. Guinea pigs sensitized by intramuscular injection of culture*

ANIMAL	INOCULATED WITH	REACTION 18TH DAY		REACTION 35TH DAY		REACTION 53RD DAY	
		JOHNIN	AVIAN TUBERCULIN	JOHNIN	AVIAN TUBERCULIN	JOHNIN	AVIAN TUBERCULIN
1	Avian T.B.	—	—	—	+	—	+
2		—	+*	—	+	—	+
3		—	+	—	+	—	+
4		—	+	—	—	—	+
5		—	+	—	+	—	+
6	Johne's B.	—	—	—	—	+	+
7		—	—	—	+	+	+
8		—	—	—	—	+	+
9		—	—	+	+	+	+
10		—	—	—	—	—	—

*A + reaction indicates that an edematous swelling, measuring at least 10 mm. in diameter, is present at the injection site at the 48th hour after injection..

evident that, in guinea pigs at least, the avian organism has greater antigenic power than the other. It is probable that the avian organism multiplied in the animal's tissues while the Johne bacillus did not. In not a single instance did johnin give a reaction in animals sensitized to the avian bacillus. On the other hand, among the animals sensitized with the Johne bacillus, avian tuberculin always gave a reaction when johnin was positive.

Before the test just described was carried out, there was no means of knowing whether the products used for testing were potent or not. The results of the test, however, suggested that the johnin used was not on a par in antigenic power with the tuberculin. At this time we happened to have several flasks of the Johne organism growing luxuriantly on a synthetic medium, so we made up a new lot of Johnin (lot XVI) and repeated the experiment on a new group of animals. Twelve animals were sensitized as before, six with each organism. Twenty-one days after inoculation, the animals were tested, the new johnin and the old tuberculin being used simultaneously, with the result that ten animals reacted to both substances, one which had been sensitized with the avian tubercle bacillus reacted to avian tuberculin but not to johnin, and one which had been sensitized with the Johne bacillus reacted to johnin but not to avian tuberculin.

We draw the following conclusions from the experiments just described:

1. Guinea pigs sensitized with the avian tubercle organism or the Johne organism will react typically to intradermal injections of either avian tuberculin or johnin.
2. If products of equal concentration are used, avian tuberculin seems to be approximately as efficient in giving reactions in guinea pigs sensitized to the organism of Johne's disease, as johnin itself. The converse is also true.

Ability of Johnin to Give Reactions in Tuberculous Chickens

For class demonstration purposes, a number of chickens had been inoculated intravenously with suspensions of the several tubercle types. Nineteen days after the time of injection, while they were yet in fairly good physical condition, these birds, together with two hens naturally infected with the disease, were given a wattle test with a concentrated johnin. The results of the test can be seen in table II.

Table II—*Wattle tests of artificially inoculated and naturally infected tuberculous birds, using johnin as the test substance*

Bird	Inoculated with	24 Hrs.	48 Hrs.	72 Hrs.
1	Avian T. B.	2X*	2X	Sl. th.
2	Avian T. B.	2X	2X	—
3	Human T. B.	Sl. th.	—	—
4	Bovine T. B.	Sl. th.	—	—
5	Avian T. B.	4X	2X	2X
6	Avian T. B.	4X	4X	1½X
7	Control	—	—	—
8	Control	—	—	—
9	Avian T. B.	2X	4X	3X
10	Avian T. B.	4X	4X	3X
11	Natural infection (tuberculosis)	2X	2X	Sl. th.
12	Natural infection (tuberculosis)	3X	4X	3X
13	Control	Sl. th.	—	—
14	Control	Sl. th.	—	—

*The figures represent multiples of the normal thickness of the wattle.

The result of this test was so surprising that an opportunity to test the product on a larger scale was sought. The opportunity soon presented itself in a flock of about 375 birds in which heavy mortality from tuberculosis had been experienced. The birds were divided roughly into two lots and one was tested with johnin, the other with avian tuberculin. Colored celluloid leg-bands were used to indicate the lot to which the bird belonged but no attempt was made to maintain the identity of particular birds. The results of the tests were read at the 24th and 48th hours. Many more reactors were present at the 48th hour and

this reading was regarded as final. Of 195 birds receiving johnin, 46 gave clear-cut reactions while 149 were negative. In the other group, consisting of 168 birds, 64 reacted and 104 did not. There was no difference in the character of the reactions in the two groups. About three-quarters of the reacting birds in each group presented swellings which were read as 3X and larger.

After an interval of fifteen days, the flock was retested, the same materials being used but the groups reversed. Several birds had died in the meantime and a few had lost their leg-bands, hence the totals on the second test do not exactly agree, but this makes little difference. Of 194 tested with avian tuberculin, 73 reacted and 121 did not. Of 166 tested with johnin, 62 reacted while 104 did not.

At the time of reading the original test, reacting birds were marked with an extra celluloid leg-band, hence it was possible on the retest to know whether individual birds had reacted on the previous test or not. This made it possible to collect the following data on the behavior of individuals.

Group I. Birds which reacted on original test with johnin:
Reacted on retest with avian tuberculin 35
Did not react on retest with avian tuberculin 6
Group II. Birds which reacted on original test with avian tuberculin:
Reacted on retest with johnin 51
Did not react on retest with johnin 13
Group III. Birds which did not react on original test with johnin:
Reacted on retest with avian tuberculin 38
Did not react on retest with avian tuberculin 115
Group IV. Birds which did not react on original test with avian tuberculin:
Reacted on retest with johnin 11
Did not react on retest with johnin 91

Summing up the data, it will be found that 137 birds had reacted to avian tuberculin and 103 to johnin. In 86 instances the birds reacted to both tests. In 51 instances birds reacted to avian tuberculin but not to johnin and in 17 instances they reacted to johnin but not to tuberculin.

Autopsies upon the birds were not obtained. We had examined a considerable number of birds dying before and during the tests and practically all of them had tuberculosis, hence we are sure that the incidence of the disease in the flock was very high.

The tuberculin and johnin used for this test were of the same lots as those used for the guinea pig tests described previously and illustrated in table I. The guinea pig experiments and these were carried on almost simultaneously and we had not yet learned that the johnin was lacking in potency. It is regretted that this was

the case. We believe that if a more potent product had been used there would have been better agreement between the two tests.

From our tests upon chickens we draw the following conclusions:

1. Tuberculous birds react in a large percentage of cases to concentrated johnin introduced into the wattle.
2. The reactions obtained are not inferior to those obtained with avian tuberculin.

Johnin and Avian Tuberculin Reactions of Sheep Artificially Infected with Johne's Disease

In November, 1926, four spring lambs were drenched with a suspension of finely chopped mucous membrane from the intestine of a cow which had died of Johne's disease. The material was very rich in bacilli. A few weeks earlier the lambs had received an intravenous injection of a pure culture of the Johne organism. At that time two additional lambs had been left as controls. When we were drenching later with the mucous membrane, a mistake in identity was made and one of the control lambs was drenched, hence we had six lambs on hand, one of which had not been subjected to infection, three had been both fed and injected, one had been injected but not fed, and one had been fed but not injected.

In March, 1927, approximately five months after the time of infection, johnin was administered intravenously. Two lots of johnin were used, one obtained from a commercial company and the other made by us. Nothing was known of the potency of the product which we had made, hence we decided to use ten cubic centimeters as a dose. The manufacturers of the commercial product advised a 5-cc dose of their product for cattle. Knowing that ordinary tuberculin, even in excessive doses, exhibits little or no toxicity for normal animals, we decided to use the dose recommended for cattle. The temperature chart of the test is shown as table III.

All of the animals were profoundly affected by the injection. Rapid respiration began within a few minutes after the injection in all cases. Two hours after injection all except sheep 6 (control) were lying down with heads extended and breathing at a very rapid rate. The control animal also was showing increased respiratory rate but was brighter than the others and moved around picking at the hay. Sheep 1, 3 and 5, which had received the product of the commercial company, appeared very much

TABLE III—*Johnin test on experimentally infected sheep*

SHEEP	8 A. M.	9 A. M.	10 A. M.	11 A. M.	12 M.	1 P. M.	2 P. M.	3 P. M.	4 P. M.	5 P. M.	6 P. M.	7 P. M.	8 P. M.	9 P. M.	10 P. M.	11 P. M.	10 A. M.
1	4.6	4.8	5.9	6.1	7.4	7.6	7.7	8.1	8.3	8.6	8.3	8.0	7.8	7.6	7.8	7.3	Dead
2	3.6	3.4	3.6	4.6	5.2	6.6	7.3	6.8	6.8	6.4	7.0	6.4	7.0	7.0	7.2	7.3	6.4
3	4.0	3.6	4.9	6.6	7.7	7.2	6.8	7.6	7.4	7.0	7.2	7.2	6.6	7.8	7.3	7.8	6.4
4	3.6	5.4	4.8	6.8	6.9	6.9	7.1	7.1	7.2	7.0	7.2	6.7	6.6	7.0	6.5	6.4	6.0
5	3.2	3.8	5.4	5.4	6.9	7.3	7.3	7.6	7.1	7.3	7.2	7.0	6.8	6.5	6.5	6.3	Dead
6	3.2	4.1	5.4	5.4	4.6	4.5	5.0	5.4	5.7	6.0	6.4	6.4	6.1	5.8	6.2	4.2	3.6

Nos. 1, 3 and 5, 5 cc commercial johnin intravenously.
Nos. 2, 4 and 6, 10 cc of our own johnin intravenously.
Injection between 9:00 and 10:00 a. m.

worse than those which had received johnin which we had made ourselves. The temperatures reached their greatest height in most cases in the early afternoon but did not go down as ordinarily happens when cattle are tested. Thirteen hours after injection, the animals were left for the night. At that time sheep 1 and 5 had developed diarrhea and appeared in very bad condition. Early the next morning these two animals were found dead. The three remaining infected animals still had high temperatures and lay around refusing to get up. It was fully a week before these animals returned to normal. The control animal appeared to be fairly normal the morning after the test and, as will be noted on the chart, the temperature was normal.

Autopsies of the two lambs which had died during the test showed a general hemorrhagic condition. Hemorrhages were present on the heart, both inside and out, on the pleura, peritoneum, in the pancreas, in the thymus but especially in the mucosa of the fourth stomach. In one case vast suggilations were present here. In the other the hemorrhages were smaller but numerous. In both cases there was edema of the lungs. In neither case were we able to find any evidence of Johne's disease although the entire intestine was examined thoroughly and a large number of microscopic preparations were examined. Inability to find evidence of the disease or the causative organism in infected sheep was also experienced by M'Fadyean, Sheather and Edwards,[11] hence we believe, in spite of our inability to demonstrate it, that the two animals had Johne's disease and that the severe reaction represented a specific reaction.

The animals which survived this test have been kept under observation constantly up to the present time, which is nearly 21 months from the time the infecting doses were given. None have ever shown the slightest sign of Johne's disease and all appear to be quite healthy.*

In July, 1927, concentrated johnin was injected intradermally into the tail folds. At 48 hours, the readings of the infected animals were th 3X, 6X and 4X respectively. The control animal was completely negative. Later in this month a second intravenous johnin test was given, using a very much smaller dose than previously. The control animal was not materially

*Since this article was written, two of the sheep have died. Sheep 3 died about 27 months after the date of infection, after scouring intermittently for about three months. It was very weak and emaciated before death occurred. Autopsy showed extensive lesions of Johne's disease and myriads of typical acid-fast bacilli were found in the diseased mucosa and in the lymph-glands of the neighborhood. Sheep 2 died about 29 months after the time of infection, after gradually losing flesh for several months. This animal was never noticed to scour. Autopsy failed to show lesions typical of Johne's disease and no acid-fast bacilli could be found.

affected by the injection and showed a maximum rise over the highest pre-injection temperature of 1.9°F. The three infected animals showed rises of 4, 4 and 4.7 degrees respectively and were depressed as before but not in the same degree as in the previous test. As before, the high temperatures persisted. Thirteen hours after the injection, they were not far from the maximum and the following morning (24 hours) they had not yet retreated to normal. In January, 1928, a third intravenous johnin test was given with about the same results as previously, although on this occasion the control animal showed a temperature rise which came within about one degree of the others. In March, 1928, a second intradermal test was administered, using concentrated johnin. The infected animals gave reactions at 48 hours which were recorded as th 2X, 6X and 4X respectively. The control again was negative.

In the latter part of May, 1928, the animals were given simultaneously intradermal injections of avian tuberculin and johnin, one being injected into each tail-fold. The products used for this test were the same as used on the guinea pigs described in table I and on the flock of tuberculous chickens. At the 24th hour after the injection, the control animal was negative at both injection sites. The first infected animal also was negative but the other two showed reactions of equal size at both injection sites. They were recorded as P4X. At the 48th hour, the reactions due to the avian tuberculin persisted and had even grown larger in one case but those due to johnin had disappeared. In this test for the first time one infected animal failed to react.

In July, 1928, an intravenous injection of avian tuberculin was given. Six cc of an unconcentrated product was used, but this was probably too much, since the control animal gave rather a sharp reaction to it. The temperature chart is given in table IV.

TABLE IV—*Avian tuberculin test on sheep experimentally infected with Johne's disease (6 cc intravenously at 8:15 to 8:30 a. m.)*

SHEEP	8 A. M.	9 A. M.	10 A. M.	11 A. M.	12 M.	1 P. M.	2 P. M.	3 P. M.	4 P. M.	5 P. M.	8 A. M.
2	2.9	4.0	5.0	5.6	7.4	7.3	7.2	6.8	6.5	6.3	7.1
3	2.8	4.8	4.6	6.1	7.0	7.6	6.2	6.5	5.9	6.6	5.7
4	2.4	3.7	4.0	5.3	6.8	7.4	7.0	6.0	6.4	6.0	6.7
6*	3.1	3.6	4.8	4.8	6.1	6.4	6.3	5.6	6.0	5.6	4.0

*Control.

Within an hour after injection, all of the animals were breathing with considerable difficulty, especially sheep 3. Within two hours,

this animal began to scour and this continued throughout the day. The control animal behaved much as in the second johnin test, when the temperature rise came within one degree of that exhibited by two of the infected animals. This animal, as in the previous tests, did not show nearly the same degree of physical reaction as was shown by the other animals. In this test, as in the johnin tests, the temperatures remained high for a long time and had not reached normal by the following morning.

From our experiments on sheep we draw the following conclusions:

1. Sheep artificially infected with Johne's disease will react equally well to johnin or avian tuberculin, whether it be administered intravenously or intradermally.
2. The reactions to intravenous injections of either johnin or avian tuberculin are very severe and prolonged.

Reciprocal Action of Avian Tuberculin and Johnin in Artificially Sensitized Calves

Five calves, varying in age from about 6 to 10 weeks, were procured from tuberculosis-free herds. An intradermal tuberculin test was negative. Two of the animals (1 and 4) were injected beneath the skin of the side of the neck with avian tubercle bacilli suspended in sterile mineral oil. Two others (2 and 3) received a similar suspension of the organism of Johne's disease. The fifth was retained as a control. Shortly after the injections and before any tests were done, one of the calves which had received the Johne bacillus suspension (No. 3) died of acute indigestion. Within a few days, firm, nearly painless swellings as large as a hen's egg appeared at the sites of injection in the other

Table V—*Tests of artificially sensitized calves*

Calf	Sensitized with	Tested with	Reaction	
			15th Day	45th Day
1	Avian tubercle bacillus	Avian tuberculin Johnin	P4X P3X	P5X P4X
2	Johne's bacillus	Avian tuberculin Johnin	Neg. Neg.	P3X P5X
4	Avian tubercle bacillus	Avian tuberculin Johnin	P2X P2X	P7X P6X
5	Control	Avian tuberculin Johnin	Neg. Neg.	Neg. Neg.

three animals. These have persisted practically unchanged up to the present time (two months).

In fifteen and forty-five days, respectively, the calves were tested simultaneously with avian tuberculin and johnin, one being injected into each of the tail-folds. The tests were read at the 72nd hour. The results are shown in Table V.

Fifty-three days after sensitization, the calves were given an intravenous injection of 10 cc of unconcentrated avian tuberculin The result is shown in table VI. As soon as a sufficient interval has elapsed, they will be given a johnin test.

TABLE VI—*Avian tuberculin test (10 cc injected intravenously at 8:30 a. m.)*

CALF	8 A. M.	9 A. M.	10 A. M.	11 A. M.	12 M.	1 P. M.	2 P. M.	3 P. M.	4 P .M.	5 P. M.
1 (Avian)	1.7	2.0	3.2	4.0	3.6	3.7	3.2	2.7	2.6	2.2
2 (Johne)	1.6	2.8	2.8	4.3	4.4	4.6	3.6	3.1	2.6	2.5
4 (Avian)	0.6	1.6	2.3	3.8	5.6	5.7	5.8	4.9	4.1	3.9
5 (Control)	1.1	2.9	2.4	2.1	2.5	2.0	2.7	2.4	2.4	2.3

The figures represent degrees above 100° Fahrenheit..

We draw the following conclusions from these experiments on calves:

1. Calves sensitized by subcutaneous injection with pure cultures of avian tubercle bacilli or with the Johne organism will react practically equally well to intradermal injections of johnin or tuberculin made from the avian tubercle bacillus.*
2. Calves inoculated subcutaneously with either the avian tubercle organism or the Johne organism quickly develop the capacity to react to avian tuberculin injected intravenously.

AVIAN TUBERCULIN AS A DIAGNOSTIC AGENT IN NATURALLY ACQUIRED AND ARTIFICIALLY INFECTED CASES OF JOHNE'S DISEASE IN ADULT COWS

For a number of months we have been using avian tuberculin in several herds, where Johne's disease exists, for the purpose of detecting infected animals which have not yet shown symptoms. In some of these herds, johnin has been used for previous tests.

*This should not be interpreted to mean that animals naturally infected with Johne's disease will react. We have done a few tests on naturally infected animals (cows) as well as on cows which were infected artificially by feeding and have never been able to get a positive skin test on them, although they generally react to the intravenous test. On another occasion we injected two animals (calves) with a culture of the Johne organism and these animals quickly became skin-sensitive. It is evident, therefore, that skin-sensitiveness is much more readily induced by subcutaneous injection than it is by the natural means of infection. With sheep the condition seems to be different, inasmuch as our animals have been skin-sensitive for a long period of time as a result of a feeding infection.

TABLE VII—*Avian tuberculin test of herd maintained for Johne's disease experiments, July, 1928. (10 cc intravenously, 7:30-8:00 a. m.)*

DESIGNATION	7 A. M.	8 A. M.	9 A. M.	10 A. M.	11 A. M.	12 M.	1 P. M.	2 P. M.	3 P. M.	4 P. M.	5 P. M.	6 P. M.	DIAGNOSIS	REMARKS
Hunt	1.2	1.7	2.0	2.4	1.9	1.8	2.2	2.4	2.8	2.7	3.2	3.2	—	Reacted to johnin once and failed repeatedly to react since
Mary	1.3	1.6	2.9	3.5	4.0	5.4	5.6	5.8	5.7	5.8	5.1	5.0	+	Natural infection. Has reacted previously
No. 8	1.4	1.8	2.4	2.0	1.9	1.6	1.5	1.6	0.9	1.6	2.0	0.8	—	Control. Has never reacted
No. 11	1.6	1.7	1.6	1.5	1.0	1.7	1.8	2.0	1.9	1.9	2.0	2.0	—	Control. Has never reacted
No. 13	1.8	1.8	1.4	2.4	2.5	2.4	2.6	3.0	3.2	3.2	3.2	3.4	—	Control. Has never reacted
No. 41	1.8	1.3	1.6	3.0	3.2	4.6	5.4	4.4	4.0	2.8	4.1	3.8	+	Year ago injected intravenously Johne's B. culture. Reacted previously
No. 42	1.5	1.7	1.4	1.6	1.6	1.8	2.7	3.5	4.2	3.2	2.3	2.8	+	Injected as above. Has not reacted previously
No. 43	1.4	1.6	1.8	1.8	1.7	2.6	3.3	4.0	4.3	3.8	3.4	2.8	+	Injected as above. Has reacted previously
No. 2	1.9	2.0	2.6	4.0	5.3	6.5	7.3	7.0	6.4	5.2	3.4	2.6	+	Fed infected tissues, Nov. 1926. Has reacted previously
No. 1	1.7	1.8	3.5	4.3	5.3	6.4	7.1	6.6	5.2	3.8	3.5	3.4	+	Fed infected tissues, Nov. 1926. Has reacted previously
No. 16	1.8	2.0	3.2	3.8	4.2	4.3	4.4	4.3	4.1	3.8	4.0	3.6	+	Control. Has not reacted previously
No. 12	1.8	1.6	1.6	2.0	2.0	1.7	2.3	2.6	3.4	3.0	3.0	1.4	—	Control. Has not reacted previously
No. 10	2.5	2.2	1.7	1.0	1.6	1.8	2.1	2.5	3.6	3.5	3.8	4.3	?	Control. Has not reacted previously
No. 7	1.5	2.0	2.2	1.7	1.8	1.8	1.8	2.2	2.4	2.7	2.6	2.0	—	Control. Has not reacted previously
No. 50339	1.7	1.0	1.4	1.6	1.6	1.4	1.4	1.5	2.2	1.6	1.9	2.0	—	Control. Has not reacted previously

Inasmuch as we are pursuing the policy of watching reacting animals, rather than destroying them, and since the majority of the animals which have reacted are still alive, we are not now in a position to make a report on the results. At the present time we have more data on a small herd which we are maintaining for our experiments than on any of the others. This herd consists of animals which have been procured as reactors from herds where the disease exists, others which were normal when brought into the herd but have been subjected to deliberate attempts to infect them, and still others which have not been especially handled but have lived in the same stable and have been pastured on the same ground with the others. Every animal in the herd has been exposed to the infection, therefore, but inasmuch as we have had no bad spreaders on the place, as far as we know, we have seen little evidence of natural spreading during the last two years. All of the animals have been tested one or more times with johnin, and one or more times with avian tuberculin. In order to give an idea of how infected animals react when injected intravenously with avian tuberculin, we are giving as table VII the record of the last test on this herd. This record includes all animals except the bull, which is segregated from the others and not known to be infected, and one clinical case of Johne's disease which we did not test because the test might have interfered with some other work which we were doing with this animal. Of the animals included in the test chart, only one, "Mary," has ever shown any symptoms of Johne's disease. This animal is emaciated but has never scoured during the eight months which she has spent in the herd. She came from a herd where Johne's disease existed and was one of a number of reactors.

From our experience with avian tuberculin as a diagnostic agent for Johne's disease, we draw the following conclusions:

1. When injected intravenously, avian tuberculin induces in cattle which are affected with Johne's disease a temperature reaction which is indistinguishable from that induced by johnin. The reaction is a sharp upward curve, which generally reaches its climax about the fifth or sixth hour after injection, after which it quickly subsides. In some animals the climax may appear somewhat earlier (second or third hour) or considerably later (twelfth hour).

DISCUSSION

In comparing solutions containing the active principle of such substances as tuberculin, we are handicapped by the lack of any

method of knowing when the active principle in one is more concentrated than in another. In the work which we have just reported, we believe that it is fairly evident that some of the lots of johnin which we have used were lacking in the potency which our avian tuberculin possessed. We have had enough experience with different lots, however, to convince us that qualitatively the principle which induces the temperature rise and other manifestations of a reaction in an animal which is allergic is very much alike or possibly identical in the two substances, and that allergic reactions can be induced with either at will. Until recently, technical details in the method of preparation made it practically impossible to obtain a johnin of much potency. In comparison with such lots we believe that avian tuberculin would be preferable as a diagnostic agent for Johne's disease. In comparison with the newer products the situation is not so clear.

Excellent growth of the specific organism can now be obtained readily and johnin made from such cultures should be rich in the active principle. These products should still be considered as in an experimental stage, however. We have, for instance, obtained some striking and perfectly typical temperature curves in calves which could not possibly have had Johne's disease or have been sensitized in any way to the avian tubercle bacillus, by injecting large amounts of johnin which we have made and also of that made by a commercial company. Others have had similar experiences. It is quite probable that the trouble here is merely that doses too large have been used. We are accustomed to think of tuberculin as a perfectly harmless substance for normal animals, even when rather large amounts are used, and so it is—when injected subcutaneously. Ordinary tuberculin is not used intravenously and it is quite possible that surprising results might be obtained if large doses were given intravenously to normal animals.

We have found, like Vallée and Rinjard,[16] that cattle may be readily sensitized to johnin (and avian tuberculin as well) by subcutaneous injection of the Johne bacillus. We have not found, however, that naturally infected cases, or cases induced by feeding, frequently become skin-sensitive. In a rather limited experience we have not found subcutaneous injection so satisfactory as the intravenous test. Reactions can be induced by this method, as many have proven besides ourselves, but they are slower in developing and the temperature increase is not apt to be so great as when the material is injected into the vein. These considera-

tions lead to the belief that animals infected with Johne's disease are not so allergic as animals affected with tuberculosis and, consequently, it is an advantage to bring the active principle of our johnin and avian tuberculin to bear on the diseased tissues as abruptly as possible, in order to obtain the best results. O. Bang used avian tuberculin subcutaneously and although he obtained fairly consistent results, his temperature charts are not nearly so striking as those of table VII. In order to detect reactors by his method it was necessary to read the reactions closely. From our limited experience with johnin injected subcutaneously, we believe that one would have to proceed similarly.

It has been pointed out frequently that avian tuberculin in the hands of M'Fadyean, Sheather and Edwards,[11] failed in many instances to give reactions in animals affected with Johne's disease. But the authors themselves recognize the fact that most of their material consisted of animals which were in advanced stages of the disease and which might not react for this reason. As a matter of fact the majority of these same animals were tested with johnin which was probably as potent as any which had been made up to that time and the results obtained were no better than those obtained with the tuberculin. In our hands both avian tuberculin and johnin have failed more often than they have succeeded, in eliciting reactions in such animals.

Schalk[13] has shown that cattle are easily sensitized so that they will react to avian tuberculin when they are associating with flocks of tuberculous birds. In this instance the sensitization is due to the ingestion of the avian tubercle organism which probably produces insignificant lesions in the digestive tract. Plum[12] has shown that a form of tuberculous metritis due to the avian type of the tubercle organism is not uncommon in cattle which are pastured on ground to which tuberculous birds have access. These animals will react to avian tuberculin though not ordinarily to ordinary tuberculin. The argument has been advanced[7] that in districts where tuberculosis of birds is fairly common, avian tuberculin as a diagnostic agent for Johne's disease would suffer from the fact that animals sensitized to the avian tubercle organism would react as well. The inference is drawn that johnin would not have this defect, but we have shown in this paper that this is not true.

Animals which suffer from tuberculosis of the ordinary type will sometimes, though not always, react to avian tuberculin. As far as published data are concerned there is little evidence that

reactions will be obtained in such animals with johnin, although it is possible that when a more potent product is used they will be observed. Thus, Turner,[14] in using one of the newer products, obtained temperature rises in two tuberculous animals which could hardly be considered other than reactions. Even if avian tuberculin should give a greater percentage of reactions in tuberculous cattle than johnin, this would not be a serious fault since it is just as desirable to eliminate animals infected with the one disease as the other.

Finally, in concluding we would like to make it clear that we are not opposed to johnin or have any idea that a product from fully developed cultures is inferior in any way, as a diagnostic substance, to avian tuberculin. Our only purpose in this communication is to show that when applied to cattle and other animals naturally or artificially infected with the organism of Johne's disease, avian tuberculin apparently has given results identical with those obtained with johnin.

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Michigan's good roads lead to Detroit.

Missouri Meeting

Dr. J. D. Ray, secretary of the Missouri Veterinary Medical Association, announces that the annual meeting this year will be held at the State Fair Grounds, Sedalia, June 25-26-27. The meeting will be held in the Veterinary Building erected at the Fair Gounds last year. Many Missouri veterinarians have never had an opportunity to see this building, toward which they contributed money for its erection. Here is an opportunity not only for Missouri veterinarians, but for members of the profession in other states, to see the first building of its kind. Dr. Ray is looking forward to a record attendance.

GLUCOSE THERAPY*

By J. H. WEINER, *Kansas City, Mo.*

In human medicine excellent results have been obtained with the intravenous injections of glucose since 1925. Its use in veterinary medicine is more recent. The introduction of glucose therapy as a routine measure in veterinary medicine was due largely to the recommendations and clinical tests made in Chicago, by Dr. L. A. Merillat and his associates, who visualized a relation between two definite diseases and the process of lactation. The one was eclampsia of nursing bitches and the other parturient eclampsia of mares. Later, when the investigations of Auger, in France; Maguire, in England; and Carlens and Widmark, in Sweden, succeeded in establishing a connection between glycemia and milk fever in cows, this disease too was added to the group. From this start, the use of glucose was extended to other states of a nervous nature associated with several more or less common animal diseases.

So that we may entirely understand one another in relation to exactly what glucose means, let me say that even though the source of supply is identical, medicinal glucose is not the glucose of commerce.

The glucose of commerce is obtained by the incomplete hydrolysis of starch and consists of some d-glucose (dextrose), dextrins, impurities and water. D-glucose, or anhydrous dextrose, or the glucose used in intravenous therapy, is prepared from chemically hydrated glucose, which is produced in a manner similar to that employed for the preparation of commercial glucose or corn sugar, except that the conversion is carried out to completion and pure dextrose is crystallized with no dextrins, semi-converted sugars or moisture present.

It is generally held by physiologists of today that all carbohydrates are converted into dextrose before they are utilized by the body cells. It is now believed that increasing the amount of this sugar in the blood supplies the working muscle a readily oxidizable, energy-yielding form of carbon, not only increasing its immediate power, but also lessens the call upon the fuel stored up. It is to this mechanism and the rapidity of its absorption

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minn., August 7-10, 1928.

that glucose owes its power of banishing the sense of fatigue caused by muscular exertion.

Dixon, in 1907, found that injections of glucose into the blood-stream may cause an immediate increase in the force of cardiac contractions, presumably by furnishing a heavy oxidizable food. Kleiner, in 1910, found that after the intravenous injection of large quantities of dextrose in dogs, the blood sugar returns to its normal quantity within one and one-half hours.

Of the excess glucose introduced, the major part is eliminated through the kidneys; about 40 per cent escapes from the blood-vessels by the process of dialysis into the surrounding tissues; and a small amount is destroyed by increased oxidization.

It has long been known that in conditions where there is an inability to oxidize blood sugar, as in diabetes, there is a tendency toward a condition commonly, though inaccurately, known as acidosis.

Chemically pure dextrose has been introduced into medicine in order to provide a form of sugar suitable for intravenous administration. For intravenous use it is essential that dextrose should be of the highest purity. Titus and Givens find that even with the best quality of dextrose it is advisable to filter and re-filter the solution because of the presence of dust particles. It is absolutely essential that the solution be completely sterilized, which may be accomplished by heating in an autoclave or regular sterilization.

In our original work on glucose we endeavored to develop a proper commercial method for the manufacture of a sterile isotonic solution but we did not meet with much success. The problem was a peculiar one, for when concentrated glucose solutions are heated to 100 degrees Centigrade, the hydrogen-ion concentration is changed markedly to the acid side and our experience taught us that injections of such acid solutions are likely to cause reactions. It became necessary, therefore, to buffer the solution, a process which is not feasible before sterilization, because the solution turns yellow, indicative of a chemical change.

Our thoughts drifted then to the use of cresol which, when added to the solution, made it unnecessary to heat it to so high a temperature for sterilization. In human intravenous medication, where dosage is small, this method temporarily sufficed, but in veterinary medicine, where large dosage and heavy concentrations

are paramount, we felt it would be an absolute necessity to do further work in an endeavor to solve this problem.

After approximately five hundred separate experiments we developed a method that gives a glucose solution of absolute sterility, water white and with no lowering of the hydrogen-ion concentration after standing for over a year. This information was given to the veterinary medical manufacturers who so desired it and from all recent reports they are obtaining the identical results we obtained in our experimental work.

On the therapeutic side the striking results which were being obtained in the treatment of bitches affected with what was now being called "lactation eclampsia" created considerable controversy as to the glycemic nature of the above-mentioned group of diseases. Attempts have been made by chemical analyses of blood to show that the analysis of the blood of animals affected with these diseases did not harmonize with the therapeutic results obtained by introducing medicinal glucose into the organism. This again brought the chemist into the foreground. Hayden, in this country, and Moussu, in France, found hyperglycemia instead of hypoglycemia in the blood of the patients they examined and for a time threw confusion into the new pathology. But owing to the fact that blood-sugar determinations, particularly when only venous blood is examined, shed no useful light upon the metabolic process of sugar utilization, it was soon realized that biological as well as chemical determinations must be considered.

For example, the quantity of total sugars which the venous blood of a cow affected with milk fever contains is no evidence of the quantity of sugar the organism is utilizing to maintain life. The reason for this is that blood sugar in the utilizable state is a highly transient product. Very little of it may be available to the organism and yet the chemist may find it in abundance. That is, glycemia in the physiological sense and in the chemical sense are two entirely different conditions. As Auger has pointed out, the fact that the blood of a cow at the time she is comatose may show an excess of sugar and that as soon as she recovers the glycemia unerringly drops to normal is proof that the art of determining utilizable sugar in the blood of the sick still lacks practical exactness.

The sum total about the controversy concerning the relations of glycemia and these diseases depends entirely upon whether the total blood-sugar calculations have any pathological meaning,

capable of practical interpretation. With their continuous changing and transient activity, the chemist regards it as impossible to give the pathologist any accurate information. Some chemists have even tried to separate glucose from lactose without first taking into consideration the selective action of the yeasts employed. In short, the first investigators took for granted that the present-day blood-sugar analysis was accurate, when in fact much remains to be learned about the differentiation of different sugars and little is indeed known about the chemistry of real utilizable blood sugar.

It is well known that when insulin is injected into the organism the blood sugar disappears and that when accidents occur therefrom injections of glucose correct the condition resulting, but it was also observed that the proper type of glucose solution was necessary to restore the normal glycemia. This led the medical chemist to attempt the production of a glucose as nearly assimilable as it is possible to produce. The therapist could not obtain curative results otherwise. Impure sugars (glucose) or sugars that are not isotonic are almost immediately eliminated and hence of no use therapeutically. The blood has no power of selecting its needs from a foreign product of this kind injected into it.

In human medicine, glucose is now injected prior to insulin. This would appear to be a paradox but for the fact that a source of energy is needed immediately to restore the activity of the sugar-starved cells, and the action of the insulin on the glucose is required to accomplish this. In the case of milk fever, for example, the normal pancreatic insulin acting upon the injected glucose brings about the same reaction. It will also be recalled that even in cases when insulin does not lower the sugar level it does cause the disappearance of glycogen from the organism, as was demonstrated by Dudley and Moraine. In their work on rabbits they showed that the liver of animals contains 5.53 per cent of glycogen; muscle, 0.57 per cent; while rabbits receiving insulin after showing convulsions and coma no more than 1.53 per cent of liver glycogen and none whatever in the muscles. When glucose is injected prior to insulin, that loss of glycogen does not occur.

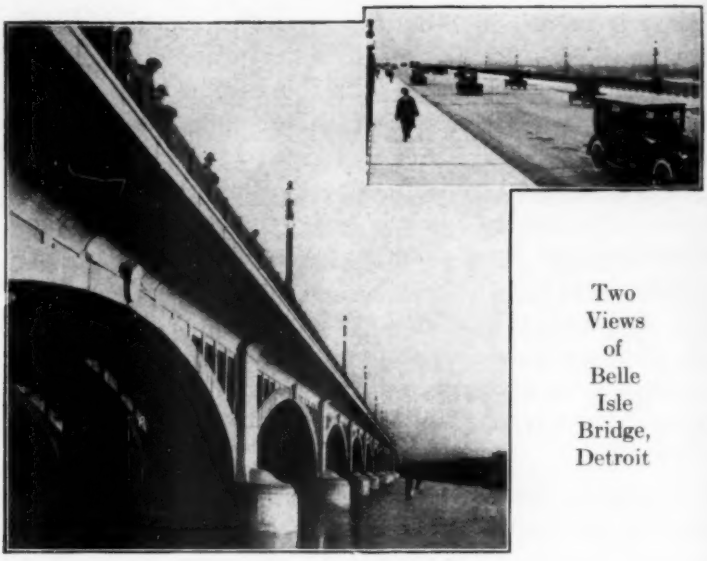
In conclusion permit me to leave this thought. Blood-sugar analyses in the case of diseases clearly due to faulty sugar metabolism are difficult to carry out to the point of fineness and

accuracy that would be of value in helping the pathologist with the method now available to the chemist, and that in judging the merits of glucose therapy the veterinarian must depend chiefly upon the clinical results obtained and that to obtain any useful results at all only a glucose manufactured with extreme care has therapeutic value.

Michigan Meeting

Dr. Edward K. Sales, secretary of the Michigan State Veterinary Medical Association, announces that there will not be the usual three-day annual meeting of the Association this year. This is on account of the A. V. M. A. convention meeting in Detroit in August. It will be necessary to hold a formal meeting, however, and June 25 has been designated as the day for the meeting, which will be purely a business session. The Capitol Veterinary Medical Association will meet at the same time and provide a short literary program.

Beautiful Belle Isle, in the Detroit River, is a paradise in August.



Two Views of Belle Isle Bridge, Detroit

FECAL EXAMINATION FOR EVIDENCE OF PARASITISM IN DOMESTIC ANIMALS*

By E. A. BENBROOK, *Ames, Iowa*

Department of Veterinary Pathology, Division of Veterinary Medicine, Iowa State College

Laboratory methods as aids to the diagnosis of animal diseases are being increasingly used by veterinarians in practice. It should be emphasized, however, that laboratory diagnosis is in no way a substitute for careful observation of symptoms, followed by deductive reasoning, leading to diagnosis whenever possible. The veterinarian, even more than the practitioner of human medicine, must depend upon keen observation of his patients.

FIG. 1. Apparatus and materials used in making fecal examinations for parasite eggs.

Laboratory *aids* may be very valuable when used with discrimination. Especially is this true in the diagnosis of parasitic diseases. For instance, the more troublesome external parasites, the mange and scab mites, are very small and the microscope is often necessary in order to determine the type present. Internal parasites, on the other hand, are hidden from view unless occasionally seen in the body discharges. Fortunately for the diagnostician, most of the internal parasites, particularly those of

*Presented at the sixty-fifth annual meeting of the American Veterinary Medical Association, Minneapolis, Minnesota, August 7-10, 1928.

the intestine, are prolific producers of microscopic eggs. Many of these eggs possess characteristics that distinguish the species, or at least the group, to which the parasite belongs. If, for instance, after clinical observation, an animal is suspected of harboring "worms," the proper therapeutic procedure depends upon the answer to the question: "Are they hookworms, round worms, tapeworms or whipworms?" The answer can usually be obtained only after an autopsy or a fecal examination; the latter method being the only practical solution where individual animals or valuable animals are under consideration.

FIG. 2. Showing how to make a "headed" glass rod by heating it in the flame and then pressing on a cold metal surface.

The method of fecal examination to be described has been in use for a number of years as a routine procedure on animals entered in the veterinary hospital at Iowa State College, and the results have been very gratifying. Particularly has this been true with horses, dogs and cats. Cattle, sheep, pigs and chickens have also been examined with somewhat less satisfactory results; due to the fact that, in the cases of cattle and sheep, relatively few internal parasites are encountered in Iowa; and, in the cases of pigs and chickens, their abundance in Iowa makes the actual finding of the parasites possible by frequent autopsies.

The method is practical, in that it takes only from 5 to 10 minutes to complete an examination and the equipment upkeep is extremely low. Not only is the method useful in diagnosis,

but it may also be utilized as a follow-up method to determine whether treatment has been effective.

In 1923, Dr. A. L. Sheather, of the Royal Veterinary College of London, originated the method which follows. It has been modified in this laboratory although the essentials have been retained.

Apparatus and materials:

Microscope of the usual medical type, giving low-power and high-power magnifications of approximately 100 and 400 diameters (fig. 1).

Centrifuge: Hand, water, steam or electric types may be used, the latter being preferable.

Microscope slides: 1 x 3 inches.

Micro cover-slips: ¾ inch square.

Several stout-walled test-tubes, size 4 x ½ inch, or centrifuge tubes of 15-cc capacity.

Several beakers of 100-cc capacity; or, ordinary drinking glasses.

Wire-gauze tea-strainer of approximately 30 meshes per inch.

Several 6-inch lengths of 5-mm. glass rod.

Wooden tongue-blades.

Fine-pointed forceps, 5 inch.

Granulated sugar.

Preliminary preparation:

1. Sugar solution should be made as follows:

Granulated sugar	1 pound
Water	12 ounces

Dissolve the sugar in the water, by immersing the bottle in hot water. Add 1 per cent phenol as a preservative.

2. To make a "headed" glass rod (fig. 2), heat one end of a six-inch length of 5-mm. glass rod until it is soft enough to be "headed" against a cold metal object. The head portion should be just slightly less in diameter than the inside of the tubes used.

To make a fecal examination.

1. Pick up at least 1 gm. of feces, using a tongue-blade and place it in sufficient water to liquefy it (fig. 3). Do not use too much water.

2. Thoroughly mix the feces with the water (fig. 4).

3. Coarse particles may be removed if necessary by straining (fig. 5).

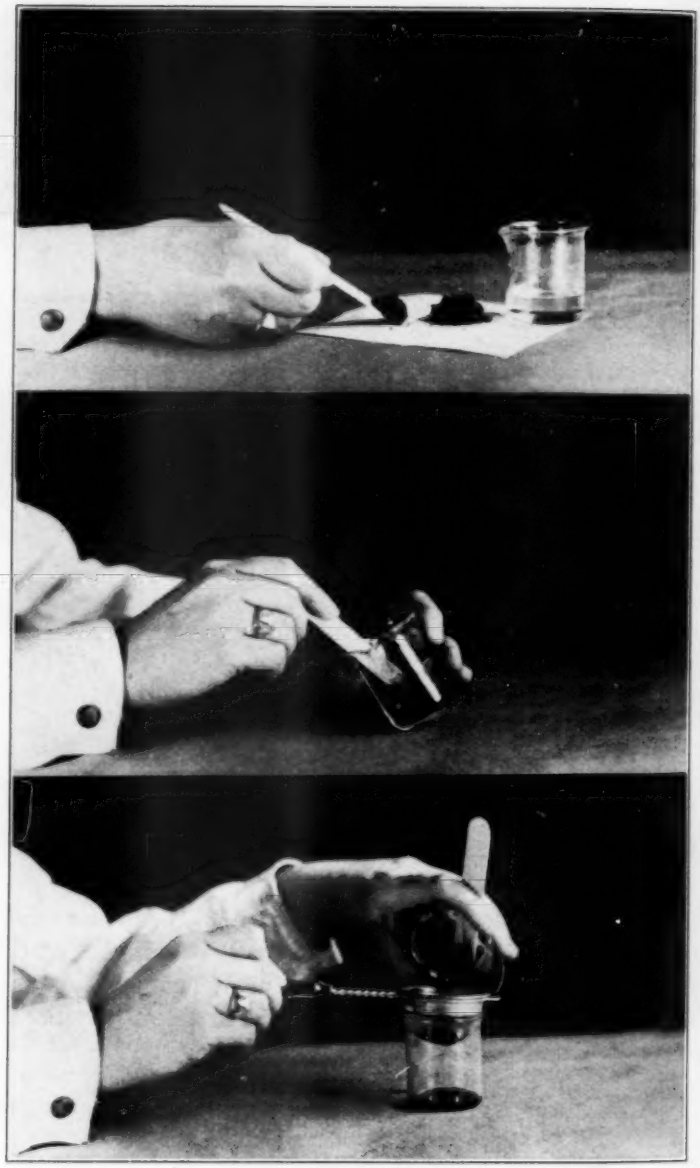


FIG. 3. (Above) Taking a sample of feces of at least 1 gram.
FIG. 4. (Center) Mixing the feces with water.
FIG. 5. (Below) Removal of course particles from feces by straining.

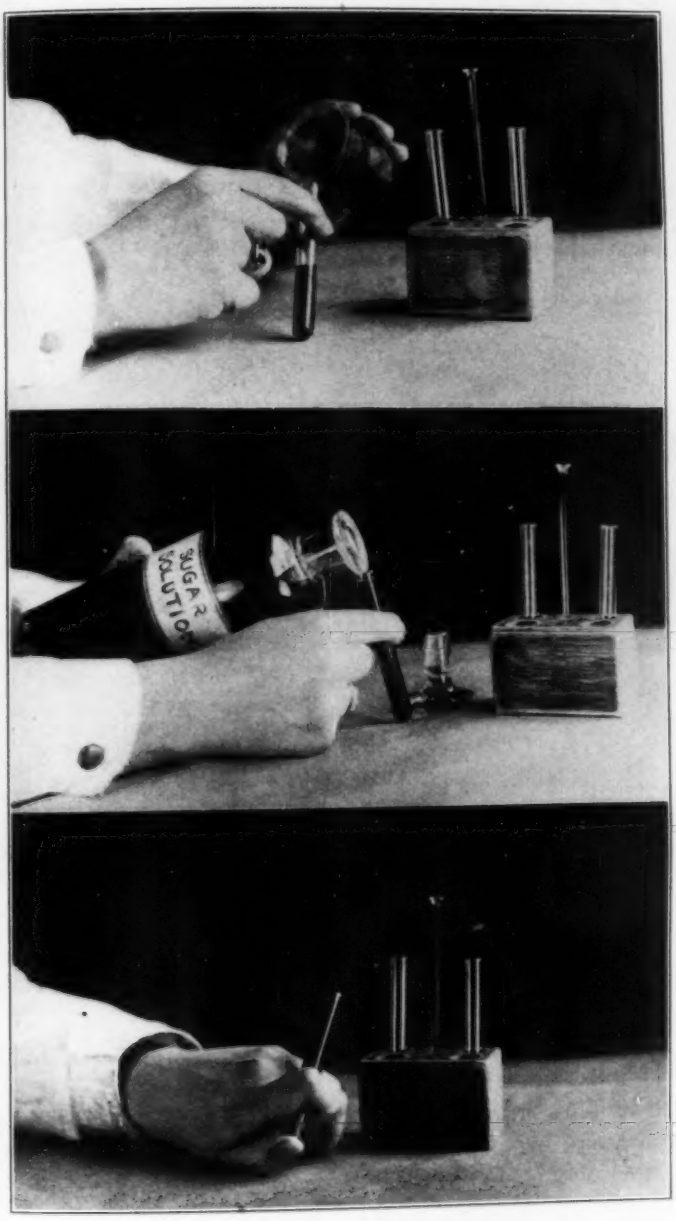


FIG. 6. (Above) Filling a tube nearly half-full of the fecal suspension.
FIG. 7. (Center) Adding sugar solution equal in amount to the fecal suspension.
FIG. 8. (Below) Mixing sugar solution and feces by inverting the tube several times.

4. Fill a test-tube or centrifuge-tube nearly half full of the fecal mixture (fig. 6).

5. Add to the above an equal quantity of sugar solution (fig. 7).

6. Mix by slowly inverting the tube several times (fig. 8).

7. Centrifuge the tube containing the mixture for about 3 minutes at moderate speed, 1500 to 2000 revolutions per minute (fig. 9). Centrifuging may be omitted if the tube is allowed to stand 12 to 24 hours.

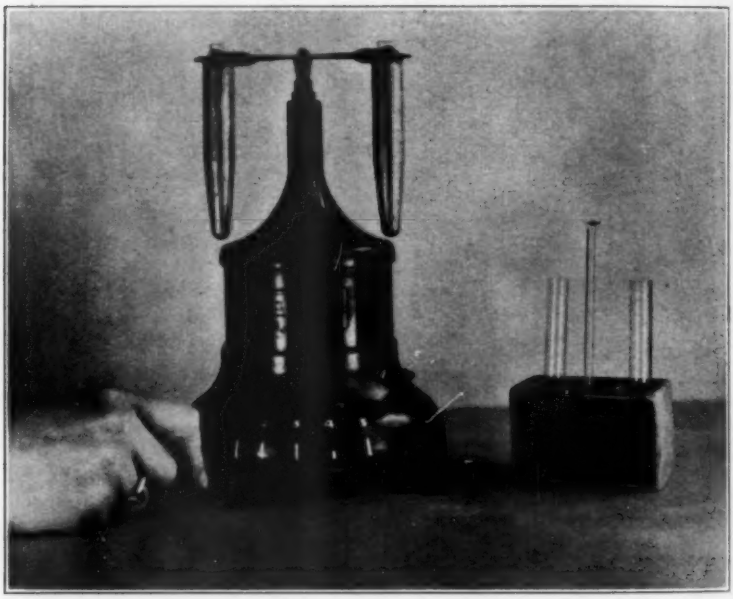


FIG. 9. Centrifuging the sample for about 3 minutes.

8. Remove the tube from the centrifuge to a test-tube holder without shaking.

9. Lift off the surface layer of fluid (which now contains the eggs) from the tube by means of the prepared glass rod (fig. 10). The rod should be slowly lowered into the tube and the instant full contact is made with the liquid withdraw the rod quickly, bringing with it a large drop.

10. Transfer the drop from the rod to a microscope slide by gently rotating the rod in the center of the slide (fig. 11). A second or third drop may be added to the first in order to obtain sufficient material to fill in under a micro cover-slip.

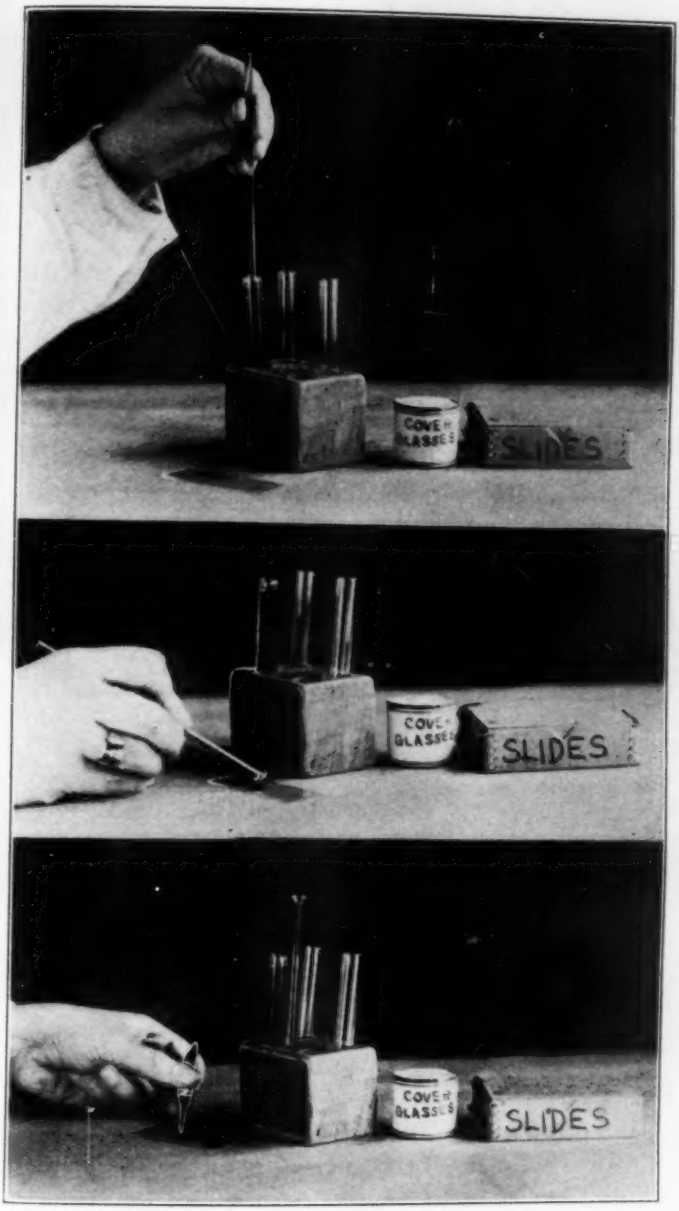


FIG. 10. (Above) Lifting off the surface layer of fluid from the tube after centrifuging, using a "headed" glass rod.

FIG. 11. (Center) Transferring a drop from the tube to a microscope slide.

FIG. 12. (Below) Applying a micro-cover-slip to the drop on the slide.

11. Carefully lower a micro cover-slip on the drop without pressure (fig. 12).

12. Examine the slide under the low power of the microscope. For best results, bright illumination should be obtained by adjusting the mirror and condenser and then modified by closing the diaphragm opening. The microscope should be vertical, not inclined, and the cover-slip area should be searched in a systematic manner (fig. 13), in order to see all parts of the drop and yet not go over the same part more than once.

13. When parasite eggs are seen, the high dry power should be used for identification in comparison with the photomicrographs that follow (figs. 14 to 31). These photographs were all taken under a microscope at magnifications of 400 diameters and will serve to identify the more common parasite eggs met with in the feces of domestic animals.

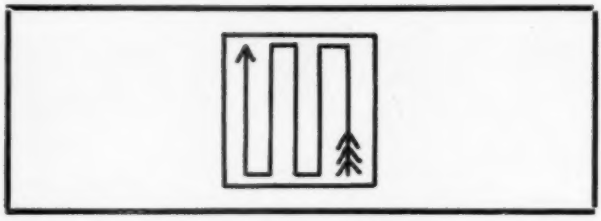


FIG. 13. Showing how the drop under the cover-slip should be systematically searched.

In closing it would be well to mention that the fecal examination method described may not be absolutely perfect in its results for various reasons:

1. No eggs may be found, because the parasites present are not mature enough to produce eggs.

2. Animals having diarrhea may pass parasite eggs so diluted by liquid feces as to make detection difficult.

3. Tapeworm eggs may be passed within the segments of the worm. Thus examination for tapeworms may be negative, or unnecessary when segments are observed in the feces.

4. Oily substances such as castor oil in the feces may interfere with the findings of eggs by floating on the top of the tube contents. Various methods have been attempted in order to remove such oil from feces but so far without success.

5. It may be confusing to find foreign eggs present. For instance, dogs may show ova of horse, pig or chicken parasites in the feces. This is no doubt the result of coprophagia.

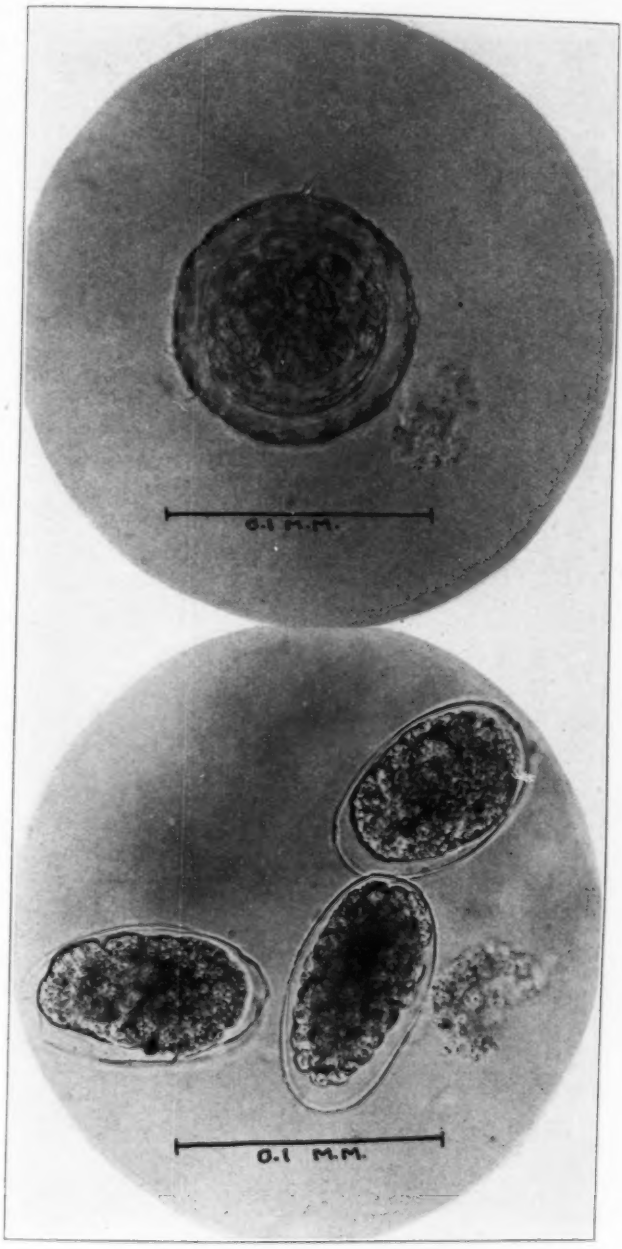


FIG. 14. (Above) Egg of *Ascaris equi*, the common roundworm of the horse. Color of egg, light yellow . x 320.

FIG. 15. (Below) Eggs of *Strongylus vulgaris* of the horse. The numerous species of *Strongylidae* family of the horse have not been differentiated by the appearance of the eggs. x 320. (See also fig. 16.)

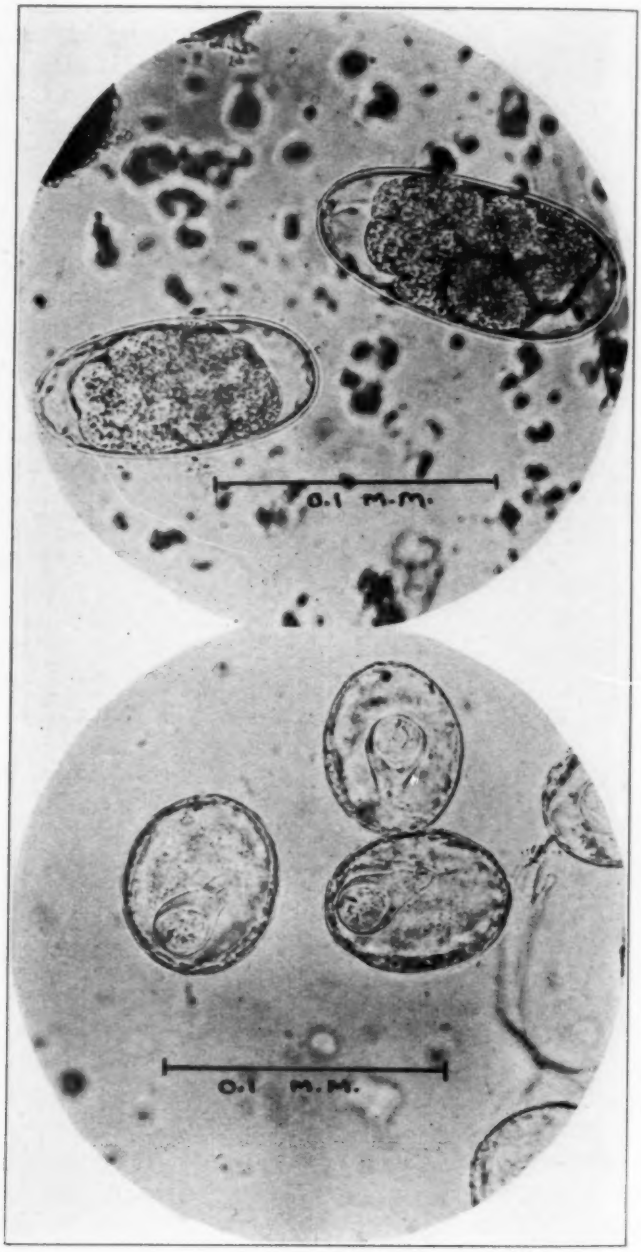


FIG. 16. (Above) *Strongylidae* eggs from the horse. Figs. 15 and 16 will serve to identify the group. x 320.

FIG. 17. (Below) Eggs of *Anoplocephala mamillana*, a tapeworm of the horse. Note the pear-shaped covering around the embryo. The other tapeworms of the horse produce similar eggs. x 320.

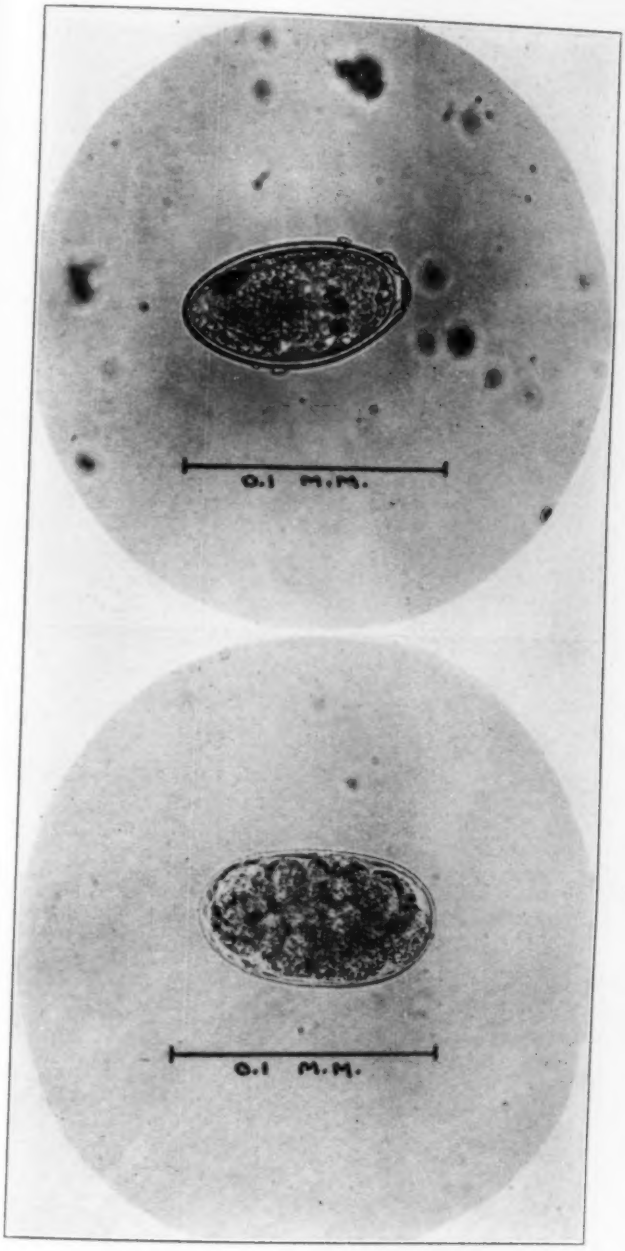


FIG. 18. (Above) Egg of *Oxyuris equi*, the pinworm of the horse. Note the operculum or cap at one end of the shell. x 320.

FIG. 19. (Below) Egg of *Haemonchus contortus*, the stomach worm of sheep, cattle and goats. x 320.

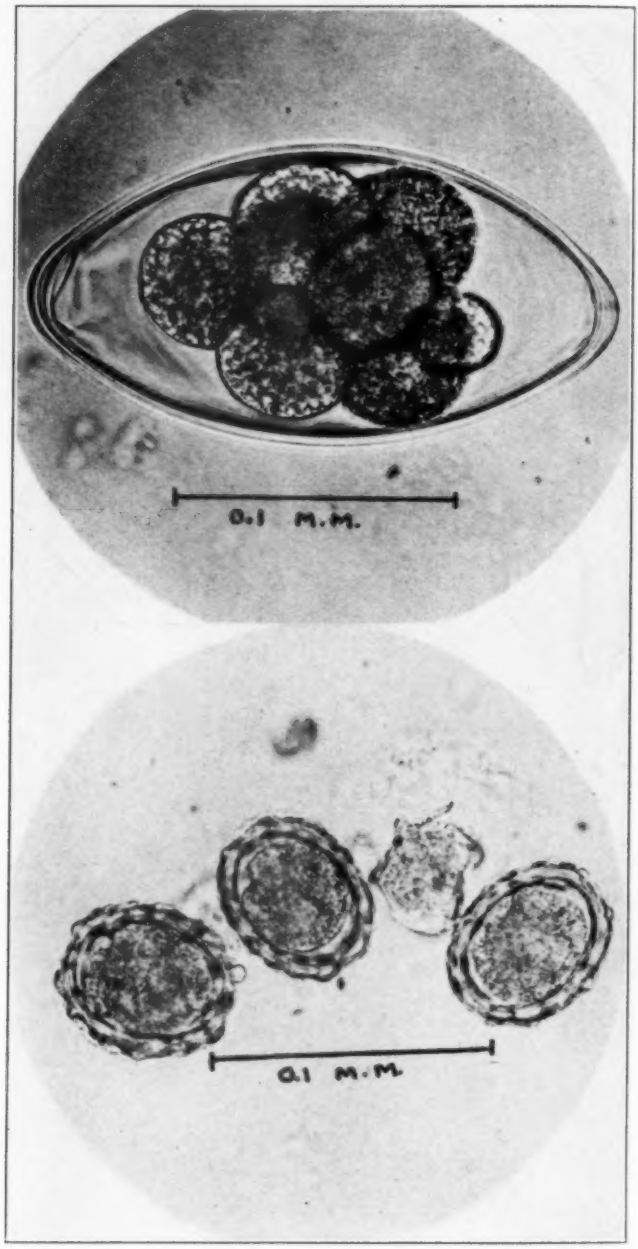


FIG. 20. (Above) Egg of *Nematodirus fillicollis*, one of the hair-like nematodes from the small intestine of sheep. x 320.

FIG. 21. (Below) Eggs of *Ascaris lumbricoides*, the common roundworm of the pig. The eggs are yellow. x 320.

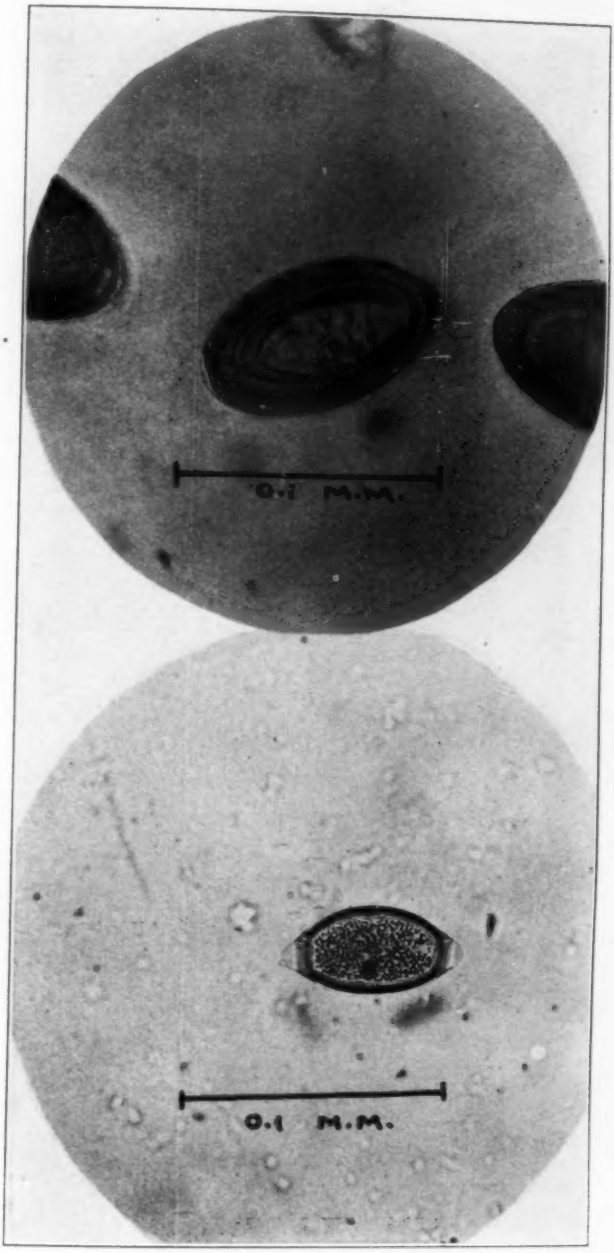


FIG. 22. (Above) Eggs of *Macrocanthorynchus hirudinaceus*, the thorny-headed worm of the pig. The eggs are brown. x 320.

FIG. 23. (Below) Eggs of *Trichuris suis*, the whipworm of the pig. Color, yellow-brown. Note the lemon-shaped outline. x 320.

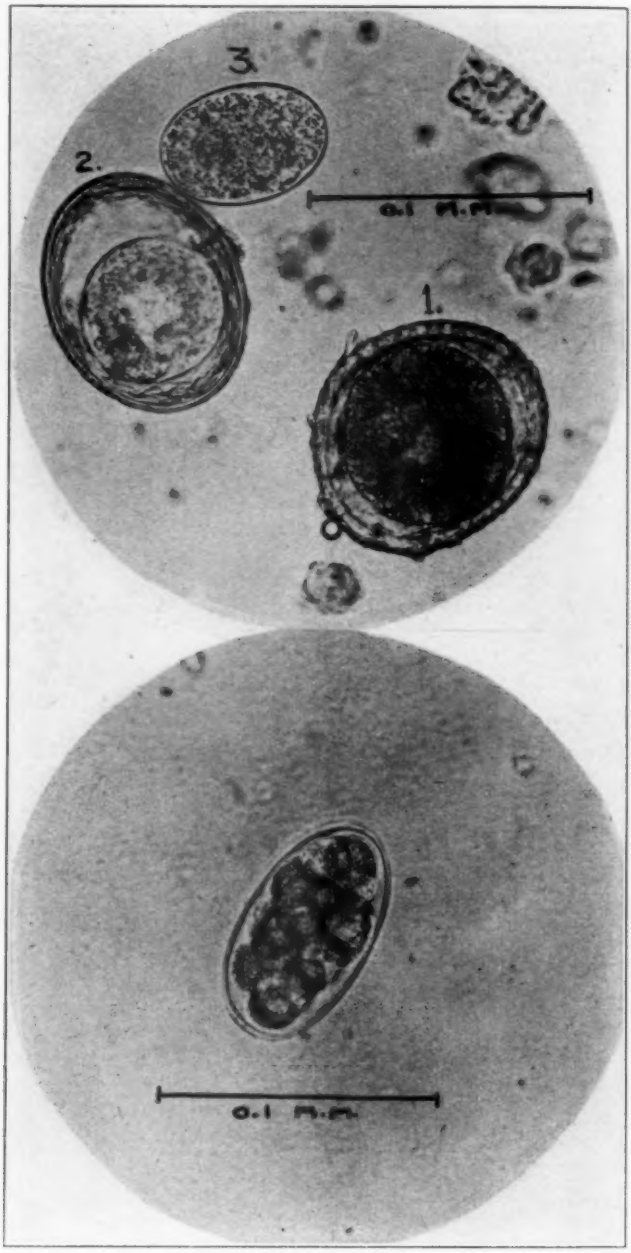


FIG. 24 (Above) Eggs from the feces of the dog. 1, *Belascaris marginata*; 2, *Toxascaris limbata*; 3, *Uncinaria stenocephala*. Nos. 1 and 2 are eggs of the common roundworms. No. 3 is the egg of a hookworm. No. 1 is yellow. Nos. 2 and 3 are colorless. x 320.

FIG. 25. (Below) Egg of *Ancylostoma caninum*, the hookworm of the dog. x 320

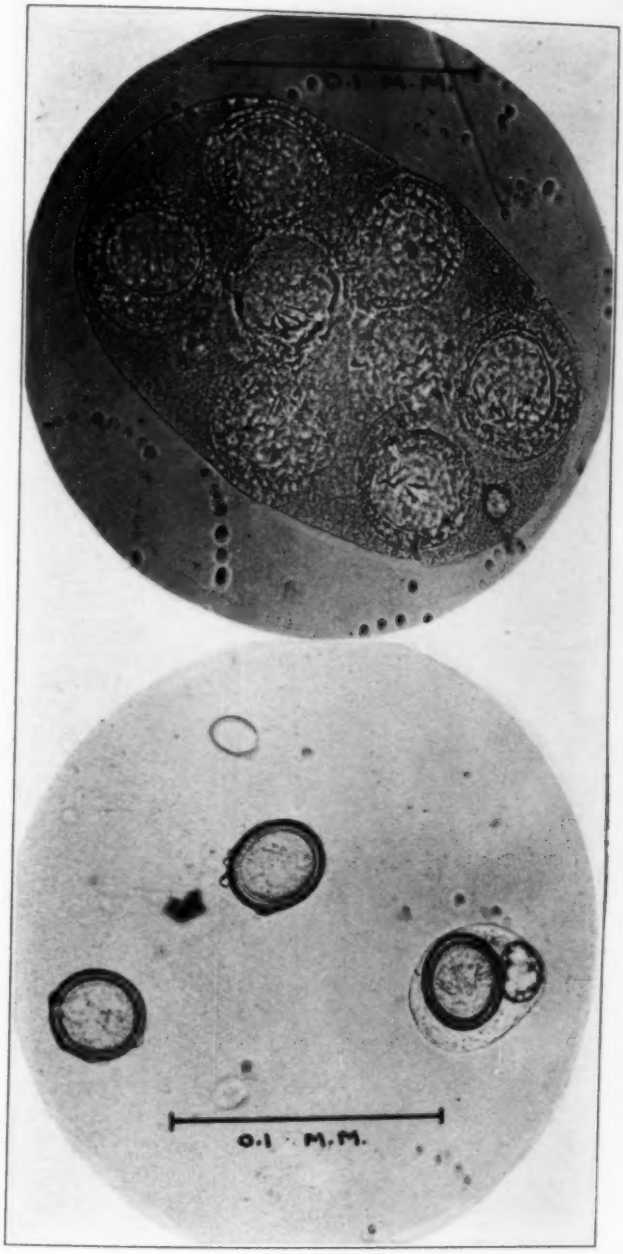


FIG. 26. (Above) Eggs of *Dipylidium caninum*, one of the tapeworms of the dog and cat. The eggs are contained in a capsule. Note the hooklets in each embryo. x 320.

FIG. 27. (Below) Eggs of *Taenia pisiformis*, one of the tapeworms of the dog. Color, yellow. Note that the upper egg is surrounded by a vitelline membrane containing a vitelline mass. x 320.

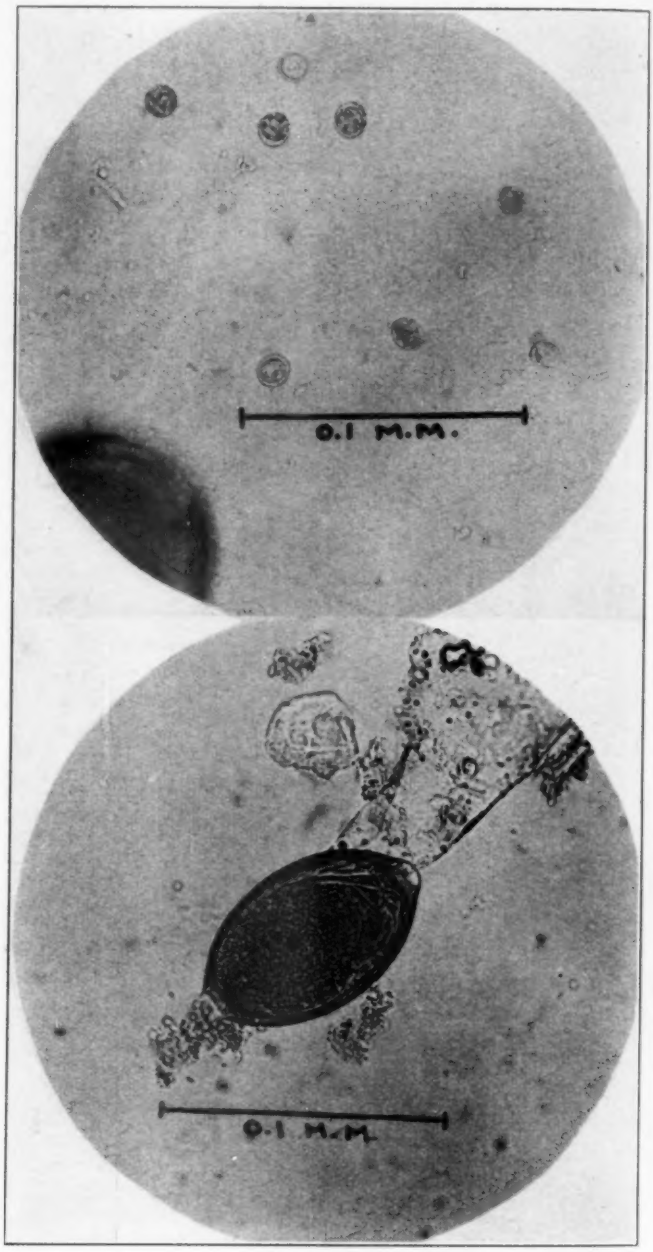


FIG. 28. (Above) Spores or oöcysts of coccidia in the feces of the dog. The dark shadow in the lower left corner is a portion of a roundworm egg. x 320.

FIG. 29. (Below) Egg of *Trichuris depressiuscula*, the whipworm of the dog. Color, yellowish brown. x 320.

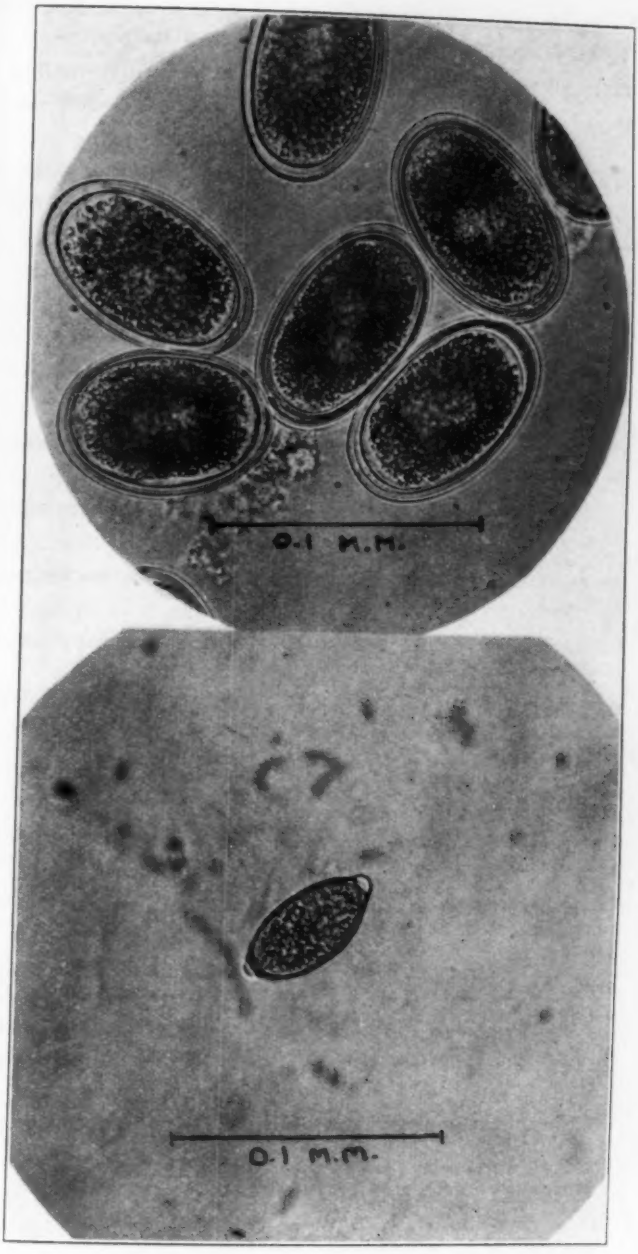


FIG. 30. (Above) Eggs of *Ascaridia perspicillum*, the roundworm of the chicken. The egg of the cecal worm is quite similar but smaller. x 320.

FIG. 31. (Below) Egg of *Capillaria retusa*, a hair-like nematode of the small intestine of the chicken. x 320.

6. Cleanliness of all utensils used is imperative, in order to prevent carrying eggs from one examination to the next. All tubes, rods, sieves and beakers should be thoroughly washed and brushed out as soon after using as possible. Wooden tongue-blades should be used only once.

It is hoped that fecal examination may be more widely used so that it will be a distinct aid to diagnostic efficiency in veterinary practice.

ACKNOWLEDGMENT

Acknowledgment is made to M. W. Sloss for assistance in the preparation of photographs.

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Michigan has the greatest coast-line of any state. The line forms at the right, in Detroit, August 13-16, led by the A. V. M. A.

INSECTS AND INSECT-BORNE DISEASES OF ANIMALS

By G. W. Rawson, *Detroit, Mich.*

Animal Industry Department, Parke, Davis and Company

While a great deal has been published on insects and their relationship to the transmission of human diseases, comparatively little information is available on the insect-borne diseases of animals. Such information as we now possess on the subject is not readily accessible, being confined, for the most part, to rather obscure reports and to text-books on parasitology in which specific information is difficult to extract from a mass of material which has little or no bearing on the subject in question.

It is the object of this paper to offer a brief review of such insects and arachnids* as are known to be capable of transmitting certain diseases to animals either in a purely mechanical manner or as intermediate hosts. No attempt has been made to include or discuss pathological conditions, such as myiasis, etc., produced as a result of the attack of insects or their larvae per se. To do so would make this paper too long.

Anthrax

Flies undoubtedly play an important role in the spread of anthrax to animals. Those chiefly concerned, of course, are the bloodsucking flies. As early as 1869, Raimbert[1] showed that guinea pigs injected with emulsions made from the various organs of flies would develop anthrax. The horse flies (*Tabanidae*) are undoubtedly the chief carriers of anthrax, as they are large and voracious blood-suckers. That horse flies do actually transmit anthrax has been definitely proved by experimental tests. Morris (1918), working on anthrax in Louisiana, proved that the horn fly *Lyperosia irritans* (*Haematobia serrata*) can transmit anthrax to guinea pigs by means of its bite. The same investigator also found the spores of anthrax in the feces of the horn fly up to six hours after feeding, in horse flies one to twelve hours after feeding and in mosquitoes 48 to 72 hours after feeding.

Aside from the transmission of anthrax by flies in a mechanical manner, it is possible also for the disease to be transmitted in a direct manner. Grayham-Smith produced infected blow flies by feeding larvae on meat infected with the spores of anthrax. It

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*A class of Arthropods represented by the spiders, mites and ticks.

was found that the blow flies remained heavily infected for two days, even after emerging from the pupal stage.

The bacillus of anthrax has also been isolated from the limbs and intestinal contents of flies more than fifteen days after they were fed on infected material. From this it is quite obvious that the secretions of infected flies could readily transmit anthrax by contaminating the drinking water or food of susceptible animals. It is perhaps not entirely fair to incrimate flies as the only insects capable of transmitting anthrax, as undoubtedly any insects

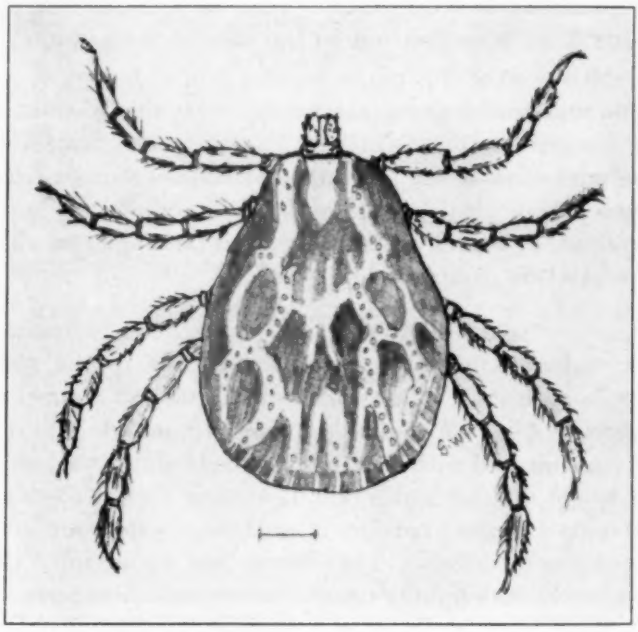

FIG. 1. The American dog or wood tick (*Dermacentor variabilis*). One of the reputed carriers of tularemia.

whose habits are suitable would be capable of spreading this virulent disease, at least in a mechanical manner.

TUBERCULOSIS

That flies are sometimes the active means of spreading human tuberculosis has been well established and there is every reason to believe that they are also instrumental in spreading tuberculosis to animals by contaminating their food or water and, in the case of poultry, through ingestion of the larvae of flesh flies that have fed on infected meat.

Schalk[2] reports an experiment in this connection of feeding non-tuberculous chickens on the larvae of flies that were feeding on the tuberculous carcasses of poultry. Of two birds fed in this way, one died three months later with marked tuberculosis of the liver, and the other died of sarcoma of the liver. While this experiment was not conclusive evidence, it shows the importance of general sanitation and the necessity for destroying the bodies of birds that have died as a result of tuberculosis.

Limberneck (Botulism)

According to Bengtson,[3] limberneck (botulism) in chickens is believed to be traceable, at least in some instances, to the ingestion of flesh fly larvae that have fed on meat infected with type C *Bacillus botulinus*. The organisms are thought to develop in the unburied bodies of dead animals, and flesh fly larvae feeding thereon become infected and in time cause the development of botulism in fowls that eat the infected maggots.

Tularemia or Deer Fly Fever

The recent outbreaks of tularemia in widely separate sections of the country have awakened much interest and have been made the theme of many a published article. Tularemia produces a plague-like disease in animals and was first reported by Francis and Mayne (1921) to be transmitted by blood-sucking insects which had previously fed on diseased rodents. The principal vectors of tularemia are the deerfly (*Chrysops discalis*), the wood tick (*Dermacentor andersoni*), the rabbit tick (*Haemaphysalis leporis-palustris*) and the rabbit louse (*Haemodipus ventricosus*).

Seven species of insects have been found capable of carrying the infection, namely, the rabbit louse, mouse louse, wood tick, bedbug, deer fly, squirrel flea and stable fly. In the deer fly, the infective agent remains alive for only a few days, but in the body of the wood tick, it remains alive throughout the lifetime of the latter. Aside from the transmission of the disease by insects, tularemia may be contracted also by contact with diseased animals. Wild rabbits and other rodents are supposed to be reservoirs of infection. Tularemia has been identified in coyotes and its transmission from rabbit to coyote has been experimentally demonstrated.

Trypanosomiasis of Animals

Since the classic discovery of David Bruce that the tsetse fly was responsible for the transmission of African sleeping sickness,

flies have been found to be of immense importance in the transmission and spread of various diseases of animals caused by trypanosomes, not merely as mechanical carriers, but as intermediate hosts upon which the infective organism depends for the completion of its life cycle, and by means of which it spreads from infected to susceptible animals. The trypanosome named in honor of Bruce, *Trypanosoma brucei*, is transmitted by the tsetse fly, *Glossina morsitans*, and causes a fatal disease in horses and mules known as nagana. It affects also cattle and sheep but is not so fatal to these animals. Nagana is one of the most important animal diseases of Africa.

Baleri, a trypanosome disease of horses, cattle, sheep and goats of the French Sudan, is transmitted by another tsetse fly, *Glossina palpalis*. A disease very much resembling nagana (caused by *Trypanosoma congolense*) also is found in the Union of South Africa and in Rhodesia, and is transmitted by *Glossina morsitans*.

Surra, a disease of horses, mules, camels, elephants and dogs, in India and in the Orient, was found by Mitzmain[4] to be transmitted by a horsefly, *Tabanus striatus*. Later, Major Kelser, of the U. S. Army Veterinary Corps, confirmed Mitzmain's observations, so that there is no question that this fly (at least in the Philippine Islands) is capable of transmitting surra from one animal to another in a mechanical manner. The stable fly, *Stomoxys calcitrans*, is thought by some to be implicated in the transmission of surra, but direct evidence to that effect does not seem to be available. It is possible, however, that other flies than Tabanids are responsible (as vectors) for the transmission of surra.

According to Darling, murrina, a trypanosome disease of mules, also is transmitted in a purely mechanical manner by flies which have fed on the skin lesions of infected animals.

The tsetse fly, *Glossina morsitans*, transmits Rhodesian sleeping sickness to humans. Animals, especially wild African game, are supposed to be important reservoirs of infection. The trypanosomes in these animals rarely produce symptoms, as the native animals of Africa have apparently adjusted themselves to this type of infection.

Another trypanosome disease of horses and camels in Algeria, namely, el debab, is evidently spread by horse flies, *Tabanus berberum*, *Tabanus nemonalis* and *Tabanus tormentosus*. No definite evidence exists that our own native trypanosome disease of horses and mules, namely, dourine, is transmitted by flies or

other insects. Apparently all attempts to transmit the disease experimentally in this way have failed. Sieber and Gonder claim to have succeeded in transmitting dourine through the medium of the stable fly, *Stomoxys calcitrans*, but this is certainly not the common method of its transmission.

The South American trypanosome disease of horses and mules, known as mal de caderas (disease of the hind-quarters), also is thought to be capable of being transmitted by flies, but no definite proof of this seems to exist.

That flies are instrumental in the mechanical spread of other diseases of animals has long been suspected. Such diseases as glanders, swamp fever, equine influenza or pink eye, etc., may possibly be spread by flies and other insects, but this needs experimental proof, so will be left for further inquiry.

Malaria of Birds

In 1898, Ross demonstrated the life cycle of bird malaria (proteosoma), showing the formation of zygotes and the presence of sporozoites in the salivary glands of mosquitoes fed on birds, while little information is apparently available on malaria of birds, the disease is thought by some authorities to have been introduced into the United States from Europe by the common English sparrow. Malaria of birds is apparently of little economic importance.

Bubonic Plague

The bubonic plague, known during the middle ages as the Black Death, is a most important disease of man.

As the *Bacillus pestis*, the etiologic factor of bubonic plague, produces a like disease of rats and other rodents, it is not out of place to mention the disease in this paper. In India the rat flea, *Xenopsylla cheopis*, is the intermediary host of the plague bacillus from rat to rat and from rat to man. In Europe and the United States, *Ceratophyllus fasciatus*, as well as other species of fleas, may transmit the disease. Bedbugs also will transmit plague. Fleas suck up the septicemic blood of infected rats and the bacilli are passed out with the feces. These being rubbed into the puncture of the flea-bite, bring about infection. Regurgitation as a result of obstruction by masses of plague bacilli in the esophagus of fleas causes the injection of bacilli into rats and man in the act of biting. This latter method of transmission is thought to be more important than the former. A plague-like disease of ground squirrles is said by McCoy[5] to be transmitted by fleas and,

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under suitable conditions, these fleas might be instrumental in transmitting bubonic plague.

Piroplasmosis

With the exception of the flies, ticks are one of the most important classes of animal parasites in transmitting various diseases of animals. In this respect the Texas fever or Southern cattle tick, *Margaropus (Boophilus) annulatus*, is of great interest, not only because of its historic position of being the first parasite found to be capable of transmitting disease, but because of its economic importance to the cattle industry. It is estimated that the loss from this tick is (or was) approximately four million dollars annually. The Texas fever tick, unlike most others, attaches itself to its host and does not drop off until fully gorged with blood and ready to deposit its eggs. Texas fever is widely distributed and is enzootic in southern Europe, Central and South America, parts of Africa, in the Philippine Islands and in the southern portion of the United States. Other species of ticks are capable of transmitting Texas fever, but none in the United States appears to be capable of doing this except *Margaropus annulatus*. In Australia, the Philippine Islands and South America, *Boophilus australis* transmits Texas fever, while in Africa the blue tick, *Boophilus decoloratus*, transmits the same or a similar disease to cattle as well as African gall-sickness. This tick is probably widely distributed.

Texas fever is not the only form of piroplasmosis of animals transmitted by ticks. Piroplasmosis of cattle in Europe is transmitted by the deer or castor bean tick, *Ixodes ricinus*, while a similar disease in South America called redwater disease is transmitted by the brown tick, *Rhipicephalus sanguineus*, and East African Coast or Rhodesian fever by *Rhipicephalus appendiculatus* and by the red tick, *Rhipicephalus evertsi*.

Piroplasmosis in other animals is also transmitted by ticks. The red tick, *Rhipicephalus evertsi*, also transmits biliary fever to horses and mules in South Africa, while the net tick, *Dermacentor reticulatus*, transmits equine piroplasmosis to horses and mules in Russia.

In the Balkan States, the sheep tick, *Rhipicephalus bursa*, transmits carceag, heartwater or ovine piroplasmosis to sheep. In dogs biliary fever or malignant jaundice, which occurs in Europe, Africa and India (and possibly in some sections of the United States), is known to be transmitted by four species of ticks,

Rhipicephalus sanguineus in India, *Dermacentor reticulatus* in France, *Haemophysalis leachi* in Africa and by *Ixodes ricinus* in Germany and Hungary. One species of flea is known also to transmit the disease, so all the blame is not to be placed on ticks. Poultry and other fowl also are victims to tick-borne diseases. In the southern states and in South America, piroplasmosis of fowls is transmitted by the chicken tick, *Argus miniatus*, and by *Argus persicus*, in Persia and Tunis.

Rocky Mountain Spotted Fever

While Rocky Mountain spotted fever is a disease which concerns man far more than the lower animals, ground squirrels,

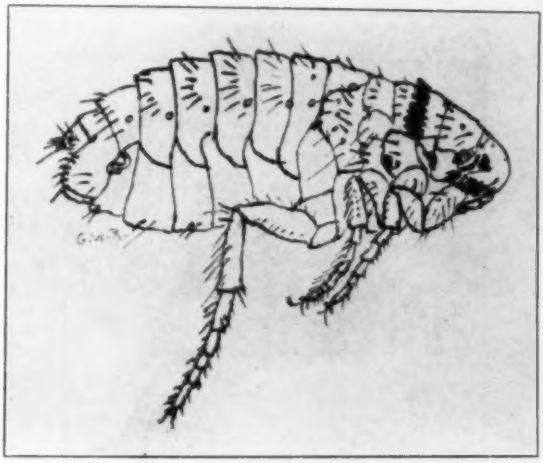

Fig. 2. The dog flea (*Ctenocephalus canis*). Female, greatly enlarged. Intermediate host of the dog tapeworm.

chipmunks, mountain rats, etc., harbor the virus of the disease, and the wood tick, *Dermacentor andersoni* (*Dermacentor venustus*), feeding upon them becomes infected and so transfers the disease to man. Spencer[6] states that one egg from an infected tick is sufficient to infect a guinea pig. Other species of ticks have been shown to be capable of transmitting the virus of Rocky Mountain spotted fever to other animals. For instance, ticks from cotton tails (*Haemaphysalis leporis-palustris*) have been proved to be capable of transmitting the disease from one rabbit to another. However, it is assumed that the wood tick, *Dermacentor andersoni*, is the species of tick responsible for the spread of the infection, at least to man.

Anaplasmosis of Cattle

Anaplasmosis caused by various protozoan parasites, principally by *Anaplasma marginale*, is transmitted by ticks in other countries, but what connection, if any, ticks have to the spread of the disease in the United States is not definitely known. Theiler[7] succeeded in transmitting the infection to cattle by the blue tick, *Boophilus decoloratus*, and by the black tick, *Rhipicephalus simus*. Helm showed that the larvae of the deer tick, *Ixodes ricinus*, hatched from ticks that had fed on infected cattle, were capable of transmitting piroplasmosis. Rosenbusch and Gonzales, in the Argentine, succeeded in demonstrating that a sheep tick, *Margaropus micropilus*, was capable of transmitting the disease when the outside temperature was high (above 34° C.).

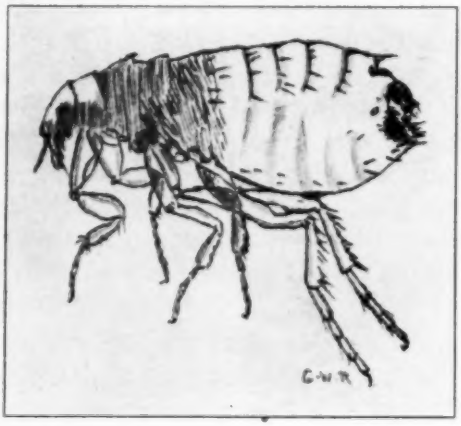

Fig. 3. The cat flea (*Ctenocephalus felis*). Greatly enlarged.

As anaplasmosis occurs in sections free from ticks, it has been suggested that biting flies such as *Tabanids* or even mosquitoes may play the rôle of vectors. Stiles[8] injected the macerated bodies of the short-nosed louse, *Hematopinus eurysternus*, obtained from cattle suffering from anaplasmosis into a guinea pig two days after removal from the host. Eleven days following the injection, a few piroplasma-like bodies were observed in the pig's red blood cells. Sixteen days after inoculation, the guinea pig's temperature was 103° F. and one to four cells per field showed coccoid bodies resembling anaplasma. About one month later these bodies had disappeared and the pig seemed normal again in every respect. Further experimental work is necessary before

we can definitely fix the responsibility of insects as vectors in the transmission of anaplasmosis to cattle in the United States.

Tick Paralysis

Fatal cases of paralysis in man, as well as in dogs and other animals, have been reported by Ross[9] on the East Coast of Australia following the bite of the tick, *Ixodes holocyclus* (female). The death rate in dogs appears to be very high. No causal organism can be demonstrated, so it is assumed that the symptoms result from some toxic principle, excreted by the salivary glands of the tick, which reaches its highest degree of toxicity two days before the female drops from her host. An emulsion of the salivary glands of these ticks proved to be toxic when injected

Fig. 4. The dog louse (*Trichodectes latus*). Greatly enlarged. Intermediate host of the dog tapeworm.

into a dog. If engorged ticks are removed many animals are said to make a complete recovery even though the symptoms of paralysis have developed.

Striking forms of paralysis due to the bite of the wood tick, *Dermacentor andersoni*, in both man and sheep (and in other animals such as dogs, foxes and rabbits), have been reported in British Columbia and in Montana. The paralysis develops gradually, beginning with a staggering gait and terminating with general paralysis.

In South Africa, tick paralysis causes considerable loss in sheep. The species of tick in this case is *Ixodes pilosus*.

Parasitic Worms

A number of insects are known to be intermediate hosts for certain parasitic worms. Much, however, is yet unknown as to

the complete life cycle of many of the parasites of wild and domestic animals. This is especially true of many of the tapeworms.

The dog tapeworm, *Dipylidium caninum*, is known to be transmitted by both the dog flea, *Ctenocephalus canis*, and the dog louse, *Trichodectes latus*. This fact has been definitely determined by careful observations. Melnikow found the cysts of the dog tapeworm in dog lice, and Grassi found them in dog fleas. Dogs apparently infest themselves by getting these parasites into their mouth while biting itching areas of the body or by the contamination of their food or water. Even puppies may become infested. This no doubt accounts for early tapeworm infestation in these animals. Parasites 2.5 cm. long have been found in a puppy ten days old and mature tapeworms in puppies one to two months old.

According to Guberlet,[10] the common house fly, *Musca domestica*, has been definitely determined to be the intermediate host of two chicken tapeworms, *Choanotaenia infundibulum* and *Davainea cesticellus*. This author states also that the stable fly, *Stomoxys calcitrans*, is the intermediate host of another chicken tapeworm, *Hymenolepis carioca*. This, however, is now doubted, as more recent work has shown that the dung beetle, *Aphodius granarius*, is an intermediate host of this parasite. Working on parasitic diseases of quail in southern Georgia, Cram[11] succeeded in infecting the ground beetle, *Anistarsus agilis*, and the dung beetle, *Choeridium histeroides*, with the cysticercoid stage of a tapeworm, *Davainea cesticillus*, found in turkeys, guinea fowls, pheasants and quail.

Flies no doubt play a very important part in the mechanical transmission of various parasites, especially while such are in the egg stage. For instance, the house fly is thought to be capable of transmitting at least six species of tapeworms and at least eight species of roundworms.

The gizzard worm, *Spiroptera hamulosa*, which is sometimes found embedded in the mucous membrane of the gizzard of domestic fowls is believed to be transmitted by the common gray sow bug. To be entomologically correct, the sow bug is not an insect, but a crustacean which flourishes under moist, rotting boards. Possibly it acts as an intermediate host. Positive evidence on this point, however, is not yet available.

A filarial worm of horses, *Habronema muscae*, was thought to be the cause of bursattee or summer sores in horses. The finding of these worms in the sores by Ercolani,[12] in 1860, and eight years

later by Rivolta,[13] gave rise to the belief that they were the etiological factor of the disease. In 1911, Ransom[14] published a short paper, which later was expanded into a bulletin, stating that *Habronema* embryos liberated in the stomach of the horse passed out with the feces and become parasites of fly maggots feeding in the manure. By comparative observations Railliet[15] formed the opinion that these larval worms were the possible cause of bursattee.

In a recent paper, Freeborn, Hart and Howell[16] have shown by careful examination that bursattee occurs without the presence of parasites, so that they are of the opinion that the nematodes demonstrated in bursattee lesions are accidental or of little etiological significance. According to Schwartz,[17] three species of stomach worms belonging to the genus *Habronema* infest the stomach of the horse, namely, *H. muscae*, *H. megastoma* and *H. microstoma*. The first two pass their larval stage in house fly larvae and the latter in larvae of the stable fly. Mention is made by Schwartz that "a condition in horses reported from various parts of the world as a granular dermatitis, or cutaneous habronemiasis, is, according to the observations of various competent investigators, due to *Habronema* larvae, probably to the larvae of all three species, although the larvae of only two species, *H. muscae* and *H. megastoma*, have been definitely incriminated as causal agents of this skin disease." Apparently some disagreement exists in regard to the significance of these worms in bursattee or summer sores, which will undoubtedly be cleared by further research.

In dogs a nematode, *Filaria sanguinolenta*, occasionally found lodged in the tumor of the esophagus and stomach, has been demonstrated by the researches of Grassi to be capable of passing through the cockroach. In the body cavity of this insect he found large cysts containing larval nematodes which when fed to dogs were found as parasites embedded in the mucosa of the esophagus when an autopsy was made two weeks after the dogs were treated. Natural infestation, therefore, is thought to take place by dogs eating roaches containing these cysts.

The manner in which dogs become infested with the heart worm, *Filaria immitis*, is not yet known, although Grassi believes it is by drinking water in which the larval worms have become liberated by the dissolution of the bodies of mosquitoes that have fed previously on infected dogs. It is reported that the embryos

of the heart worm swarm in the general circulation of infested dogs. These embryos have a periodicity of occurrence similar to that of the filarial worm causing elephantiasis of man in the tropics, being present in the peripheral circulation only at night. Blood-sucking mosquitoes feeding on infested dogs at night, therefore, would take up many embryos, which when liberated in water by the dissolution of the mosquitoes' bodies, might readily infest other dogs. However, there is a possibility that the larval filaria may be directly introduced by the bite of an infested mosquito. Quite a few cases of heart worm infestation in dogs have been reported occurring in Florida and other southern states, so that it would appear that this parasite is becoming more prevalent than formerly.

Investigations made by Ransom and Hall have demonstrated that dung beetles (Scarabaeidae) and the croton bug or cockroach, *Phyllodroma germanica*, act as intermediate hosts of the filarial worm, *Gongylonema scutata*, which inhabits the mucosa of the esophagus of cattle and sheep. Dung beetles and cockroaches became infested with the encysted larval stage of this worm when eggs were fed to them, so that evidence is thus furnished that animals become infested by swallowing insects bearing the encysted larvae.

It has recently been discovered that two stomach worms of hogs, *Arduenna strongylina* and *Physocephalus sexalatus*, are transmitted by a dung beetle, *Onthophagus hecate*. According to Raffensperger,[18] infestation probably takes place when beetles harboring the cysts of the worms are eaten by hogs while on pasture.

Another beetle, the cockchafer (Lachnosterna) is reported to be the intermediate host of the thorn-headed worm of swine, *Giganorhynchus hirudinaceus* (*Echinorhynchus gigas*). The eggs of this worm are eaten by the larvae of the cockchafer and, after hatching, burrow into the muscular tissue and become encysted there. As soon as such infested larvae are eaten by hogs (of which they are very fond) the encysted thornheaded worm attaches itself to the mucous membrane of the intestine and there develops to maturity. It is likely that the infestation of hogs also results from the ingestion of adult beetles.

Ransom and Hall thought the croton bug, *Blatta orientalis*, might serve as an intermediate host for a filarial worm of sheep,

Gongylonema scutata, although the *Aphodius* beetle is the usual carrier.

Recent investigation by Sanders[19] has shown that a cockroach, *Leucophaea surinamensis*, having a wide range in the southern United States and subtropical countries, is the intermediate host of Manson's eye worm, *Oxyspirura mansoni*. Infestation of the eyes of chickens occurred twenty minutes after infected cockroaches were fed to birds experimentally.

Dragon-flies have recently been described as intermediate hosts of a newly discovered species of fluke, *Prosthogonimus* sp., inhabiting the oviduct of poultry. Kotlan and Chandler[20] report finding the encysted larval flukes (Metacercariae) in the nymphs or larval form of certain species of dragon-flies; also in adult insects. Experiments were made in which birds known to be free from flukes were fed infected dragon-fly larvae, after which they were destroyed and the oviducts carefully examined. Most of the birds so fed showed numerous flukes. Both Chandler and Kotlan thought that two species of flukes might be transmitted by dragon-fly larvae, as two different kinds of cysts were found in dragon-flies.

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HETEROTOPIC DEPOSITS OF THYROID TISSUE IN A DOG ASSOCIATED WITH CARCINOMA OF THE LUNGS*

By WILLIAM H. FELDMAN

Division of Experimental Surgery and Pathology, The Mayo Foundation, Rochester, Minnesota

Accessory thyroid glands are observed not infrequently in the dog, and Sisson[1] remarked that three or four accessory glands may be present on each side, "as well as a median one near the hyoid bone." Davis[2] has recently drawn attention to the presence of deposits of thyroid tissue in the periaortic fat bodies of many adult dogs. He reported the finding of these deposits in twenty-two of fifty dogs examined and noted that those dogs which had enlarged thyroid glands also possessed hypertrophied intrapericardial thyroid bodies. Although periaortic fat bodies are present in the cat, hog, and sheep and are scantily present in the ox, Davis failed to find thyroid deposits in this tissue in the examination of twelve cats, fifteen hogs, fifteen sheep and fifteen cattle. Likewise none was found in the relatively small number of rats, mice, guinea pigs and rabbits examined.

Since it is important to recognize the possible presence of aberrant deposits of thyroid tissue in experiments involving thyroidectomy, an instance is here reported in which a relatively large deposit of thyroid tissue was found in the substance of the lung of a dog.

REPORT OF A CASE

The animal was an old male mongrel bulldog, in good condition and weighing 17.4 kg. The routine postmortem examination revealed a goodly portion of both lungs to be pneumonic; this condition probably was responsible for the animal's death. With the exception of the features about to be described, nothing of pathologic significance was noted in the examination of the body.

A large, irregular mass about 6 cm. in length by 3.5 cm. thick was found under the skin and in contact with the muscles of the ventral region of the larynx. This mass was of compact structure, and when it was incised it was noted to contain a dirty yellowish-gray gelatinous substance. Both lateral thyroid glands were markedly enlarged. The right gland weighed 27 gm. and measured 6.8 by 3.2 by 2 cm. The left thyroid gland weighed 49.5 gm. and was 7 by 3.5 by 3 cm. Both presented a somewhat mottled appearance when cut into and much gelatinous material was apparent.

Buried in the periaortic fat was a flattened, oval body of a grayish-flesh color which measured 1 by 0.5 cm. In the apical lobe of the left lung were two grayish-white nodules; one measured 2 cm. in diameter and was situated about 4 cm. from the periphery of the lung; the other, which measured 3 cm. in diameter, involved the tissue adjacent to the edge of the lung (fig. 1). The contents of the first-mentioned nodule were of a

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cheesy consistence and simulated, to some extent, the contents of a tuberculous nodule. Smears of this material, stained with Ziehl-Neelson's carbol-fuchsin method, failed to reveal acid-fast organisms. The second nodule was not so well circumscribed as the first and was of firmer consistence. Its interior presented a variable appearance; there were large areas of a gelatinous substance interspersed with areas of grayish-white tissue.

Pathologic histology: Sections obtained from the mass in the sublaryngeal region had a characteristic thyroid structure. The architecture was variable in the different areas examined and it was easy to follow all gradations from the type of a colloid goiter to definite adenomatous change (fig. 2). There was some tendency toward interstitial fibrous overgrowth such as occasionally is seen in fibrous thyroiditis.

FIG. 1. Aberrant nodule of thyroid tissue in apical lobe of the left lung.

The lateral thyroid glands presented the usual picture of colloid goiter, with considerable tendency toward adenomatous neoplasia. In many areas, the cellular overgrowth was so marked and displayed such slight differentiation as strongly to suggest the possibility of the presence of true carcinoma. Calcification was not apparent.

The nodule from the periaortic fat had typical thyroid structure, not unlike the average non-enlarged thyroid gland of the dog (fig. 3). Although an abundance of colloid material was present, there was no evidence of hypersecretion of this substance. At the periphery of the thyroid structure considerable monocytic reaction was apparent. Many of these cells were exerting phagocytic action on a melanin-like pigment which was present in some quantity.

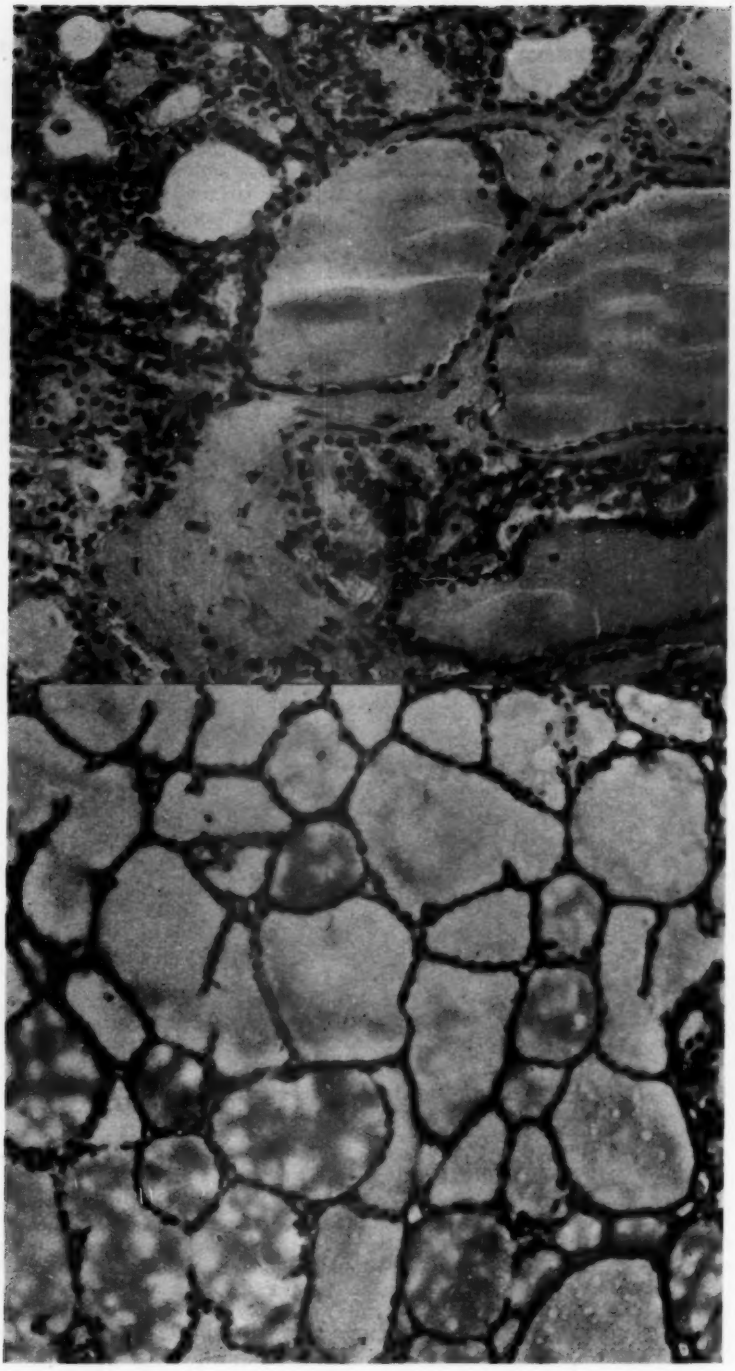

FIG. 2 (Above) Aberrant thyroid tissue from sublaryngeal region. Many of the alveoli are dilated and adenomatous changes are apparent (x220).

FIG. 3. (Below) Aberrant thyroid tissue from the periaortic fat body. The alveoli are filled with colloid (x220).

Sections of the nodule, with the cheesy contents from the lungs, showed its structure to be that of an invasive type of epithelial neoplasm (fig. 4). The manner of growth was that of an adenocarcinoma with papilliferous tendencies, and whereas the activity of the tumor was definitely localized, encapsulating elements were missing. Some areas of the tumor showed large amounts of a mucoid substance, the staining reactions of which were unlike those for the colloid material of the thyroid gland. This material apparently was the product of the columnar cells which constituted the parenchyma of the neoplastic process. Mitosis was commonly observed, and small deposits of calcium were present in some portions of the tumor.

The second nodule from the lung was typically thyroid in nature with many of the same adenomatous changes that had been observed in sections from the lateral thyroid glands (fig. 5). In no way did the structure resemble a metastatic carcinomatous deposit such as one would expect from a primary carcinoma of the thyroid gland. The greater portion of the tissue consisted of thyroid-like alveoli filled with colloid material, with a tendency toward adenomatous overgrowth appearing promiscuously throughout the tissue.

Comment

This case offers two main features of interest: first, generous distribution of thyroid tissue without the impetus of neoplasia; second, the presence in the lung of malignant epithelial tumor quite separate and disassociated from the deposits of thyroid tissue.

The thyroid tissue in the region of the larynx can be accounted for as having arisen from a remnant of the thyroglossal duct. Such a remnant may, according to Jackson,[3] in later life undergo development. The thyroid tissue in the periaortic fat and in the lung must have originated as the result of an early developing caudal extension from the main thyroid mass.

The finding of tissue of thyroid origin within the lung makes it necessary to eliminate neoplasia in the consideration of its histogenesis. In this instance, carcinomatous proliferation, although suggested, was not definitely demonstrated in any of the extra thoracic masses; further, metastatic thyroid carcinoma assumes a rather typical form in the lungs of dogs. Both lungs are involved by multiple nodules scattered quite generally throughout the organs. The nodules are of a compact consistence and, on section, reveal the adenocarcinomatous structure of thyroid carcinoma; colloid material is likely to be absent, or, if present, only in a minimal amount. In this case, the deposit was in the nature of a solitary nodule and was lacking in characteristics of a malignant tumor. It is difficult to imagine it as having had other than a congenital origin, quite unrelated to neoplasia. The presence of the thyroid deposit in the periaortic fat would strengthen, somewhat, the developmental hypothesis in explaining the deposit in the lung. The fact that the periaortic fat contained such tissue

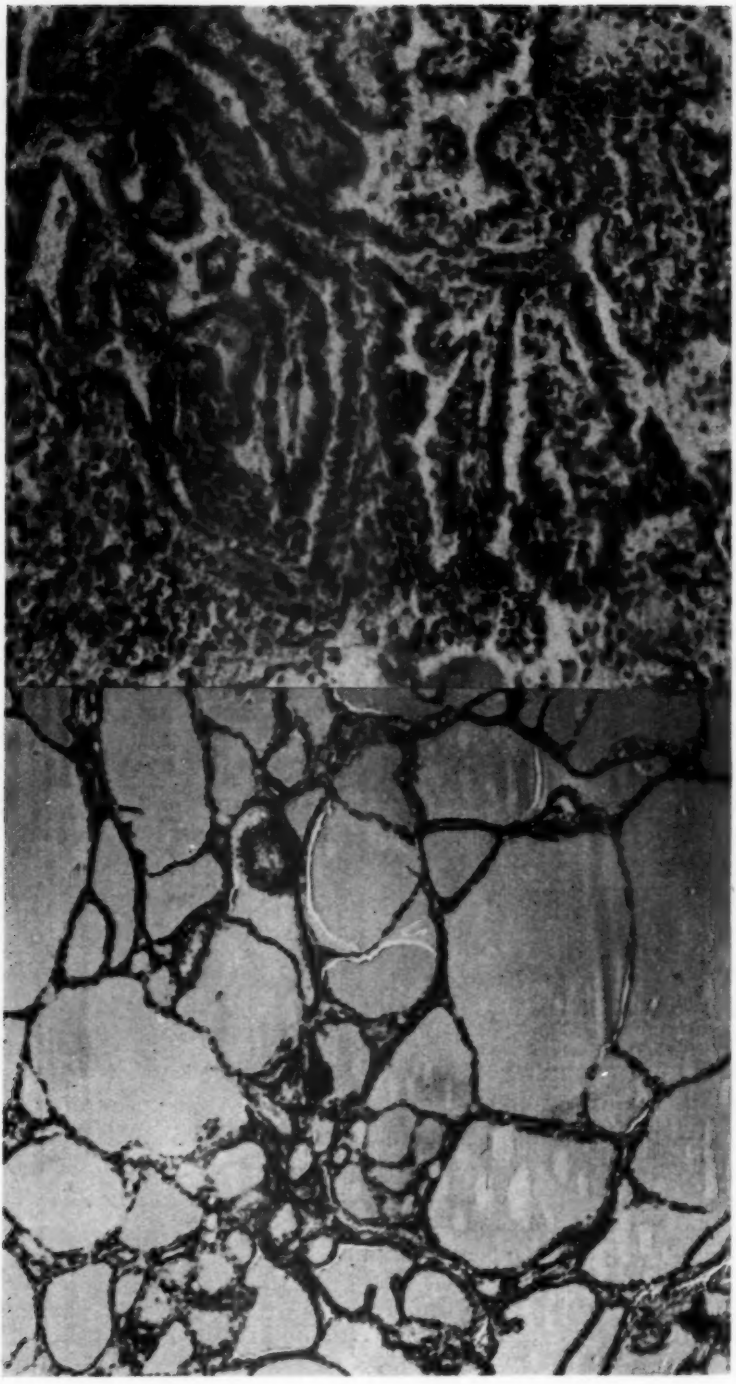

FIG. 4. (Above) Adenocarcinoma of the lung. The papilliferous character of the growth is shown (x150).

FIG. 5. (Below) Aberrant thyroid tissue from the pulmonary nodule which is shown in gross in figure 1 (x125).

should constitute tenable evidence that a developmental mishap had occurred in this animal; and since such deposits are found in many dogs in which not the slightest suggestion of neoplastic thyroid tissue is present, it would seem that the presence of the deposit in the lung could be explained by the same process.

The histologic aspect of the tumor in the lung strongly suggests its origin from the bronchial epithelium. It shows a constant preference for a papillary form of growth and does not resemble the architecture usually exhibited by a metastatic thyroid carcinoma. Further evidence against this being a metastatic expression of a thyroid carcinoma is the fact that it was limited to a solitary focus, whereas metastatic thyroid carcinoma in the dog's lung usually is characterized by the multiplicity of the nodules. The staining reaction of the mucoid material was quite unlike that for the colloid material of the thyroid gland, further emphasizing the cellular difference.

One other possibility which must be considered is the development of the tumor from an aberrant deposit of thyroid cells which may have been part of the same process of displacement; this would account for the pulmonary nodule being of definite thyroid tissue. Leech, Smith and Clute[4] have shown that in the human being papilliferous carcinomas may develop from lateral aberrant thyroid tissue. These tumors are quite dissimilar to the usual solid type of malignant adenomas which metastasize to the lungs. In a general way, the pulmonary tumor in this case resembles in some respects the tumors described by Leech, Smith and Clute. There are, however, several differences, the most important of which are the absence of any tendency toward encapsulation and the absence of colloid material as determined by staining methods. Although definite proof of the origin of the tumorous cells is impossible to present, the data obtained from the histologic study favor a bronchial histogenesis.

Summary and Conclusion

The observations at necropsy on a dog with excessive deposits of thyroid tissue are reported. The most striking features were a mass in the region of the larynx, a nodule in the periaortic fat and definite deposition of thyroid tissue in the apical lobe of the left lung. A smaller pulmonary nodule, of cheesy consistence, proved to be a papillary type of adenocarcinoma, which perhaps originated from the bronchial epithelium. The susceptibility of the dog to aberrant deposits of thyroid tissue should be considered

when this animal is used in experiments requiring complete thyroidectomy.

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Pennsylvania Alumni Day

Graduates of the University of Pennsylvania will be glad to learn that the railroads have agreed to grant convention rates, on the so-called certificate plan, amounting to a reduction of 25 per cent on a full round-trip fare, for alumni and members of their families coming from all points east of the Mississippi River to attend the Alumni Day celebrations this month. The plan is contingent upon a minimum of 150 certificates being deposited on the arrival of alumni in Philadelphia. This is substantially the same plan that has been in effect at a number of A. V. M. A. conventions during recent years. If you are going to Philadelphia for Alumni Day, be sure to ask for a certificate when you purchase your ticket.

Detroit is very close to the center of the veterinary population of North America

Michigan Central Terminal, Detroit

THE SPREAD AND CONTROL OF RABIES

By THOMAS G. HULL, *Springfield, Ill.*

Chief, Division of Laboratories, Illinois Department of Public Health

During the year October, 1927, to October, 1928, more than 1200 rabid dogs were encountered in Illinois. Through the cooperation of the Chicago Health Department and the Department of Animal Pathology of the University of Illinois, with the various laboratories of the State Department of Public Health, histories were obtained of many of these by sending a blank form to be filled in by the person submitting the head for examination.

1. Kind of animal (dog, cat, cow, horse or what)?
2. Date of first noticeable symptoms (if known).
3. Date animal was killed or died.
4. Had this animal been under observation of a veterinarian during its illness?
5. If a dog, was it a stray dog or was its owner known?
6. How many other dogs was this dog known to have bitten?
   What other animals?
   How many people?
7. Remarks (use other sheets if necessary).
8. Was dog vaccinated? If so, when? By whom?

Dogs vaccinated against rabies: The total number of dogs vaccinated against rabies in Illinois is not known. Eichhorn estimated in 1928 that not less than two million dogs in the United States had received such prophylactic treatment since 1924. Divided on the basis of the human population, this would give Illinois about 125,000 protected dogs. The total dog population is not known but is many times the above figure.

The vaccinal status of dogs examined in the laboratory was obtained on 452 rabid dogs and 86 dogs which proved to be not rabid. Among these, only one of the non-rabid dogs had been vaccinated (about 1 per cent) while 18 of the rabid dogs had received such treatment (4 per cent).

The dogs which were vaccinated and subsequently developed the disease are listed in table I. Ten of the eighteen developed the disease within one month after vaccination and all but three within two months after vaccination. It is apparent that many of them were in the incubation period when vaccinated. The effect of the vaccine on prolonging the incubation period is difficult to estimate. While periods of three and one-half months, four months and six months would indicate a failure of the vaccine, it is possible that the animals were in the incubation

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stage when vaccinated, and the incubation period was prolonged by such treatment. The possibility of the spread of rabies at dog pounds must not be overlooked. Several of the animals listed in the table had been picked up by the police and sent to the pound, to be redeemed later by the owners, only to develop rabies in three to five weeks. The one administration of anti-rabic vaccine received when the dogs were released from the pound was of course insufficient to protect them against an earlier infection.

TABLE I—*Dogs vaccinated against rabies which subsequently developed the disease*

DOG	VACCINATED	FIRST SYMPTOMS	TIME ELAPSED
1	April 9, 1928	April 9, 1928	
2	Aug. 9, 1928	Aug. 10, 1928	1 day
3	June 16, 1928	June 20, 1928	4 days
4	June 1, 1928	June 14, 1928	2 weeks
5	May 13, 1928	May 27, 1928	2 weeks
6	March 14, 1928	April 6, 1928	3 weeks
7	May 29, 1928	June 16, 1928	3 weeks
8	March 2, 1928	March 29, 1928	4 weeks
9	May 8, 1928	June 7, 1928	4 weeks
10	Feb. 25, 1928	March 23, 1928	4 weeks
11	July 15, 1928	Aug. 21, 1928	5 weeks
12	July 6, 1928	Aug. 20, 1928	6 weeks
13	June 28, 1928	Aug. 10, 1928	6 weeks
14	Feb. 9, 1928	Apr. 2, 1928	8 weeks
15	Dec. 21, 1927	April 6, 1928	3½ months
16	May 1928	Sept. 1928	4 months
17	"6 months ago"	May 6, 1928	6 months
18	Time not given	Sept. 9, 1928	

Ravages of 630 rabid dogs: The total number of dogs upon which some history was obtained was 630. One hundred eighty-six of these, listed as "stray dogs" (that is, the owners were not known), were reported to have bitten 236 people, 210 other dogs and 68 other animals. Four hundred forty-four, listed as "home dogs" (that is, the owners were known), were reported to have bitten 408 persons, 236 other dogs and 78 other animals (table II).

TABLE II—*Ravages of 630 rabid dogs in Illinois*

CLASSIFICATION	TOTAL	PERSONS BITTEN	OTHER DOGS BITTEN	OTHER ANIMALS BITTEN
Stray dogs	186	236	210	68
Home dogs	444	408	236	78

Some of the rabid dogs apparently did no damage, according to the history sheets. Others bit numerous persons, dogs and other

animals. The greatest number of people bitten by one dog was 10; of dogs bitten by one dog, 12; and of sheep and cattle bitten by one dog, 29.

Veterinarians observed 214 of the 444 home dogs for at least a part of their rabid careers. Such animals of course did some damage before their confinement, but much less than those which were not confined and allowed their freedom for the entire time. A few of the stray dogs were picked up and observed for symptoms—mostly at the pound in Chicago (see table III).

TABLE III—*Rabid dogs under observation of veterinarian*

CLASSIFICATION	TOTAL	UNDER OBSERVATION	PER CENT
Stray dogs	186	15	8.1
Home dogs	444	214	48.1

The only other animal beside the dog which was responsible to any extent in transmitting rabies was the cat. During the twelve-month period, 22 rabid cats bit 30 people. There is no indication, however, that cats were instrumental in perpetuating the disease either among themselves or other animals.

The role of the stray dog in the spread of rabies: The stray dog is the reservoir of infection in the perpetuation of rabies. The 186 stray dogs were known to have bitten 210 other dogs (a rate per hundred of 113) and probably many times this figure, for the wanderings of a rabid dog are impossible to trace. On the other hand, the 444 home dogs bit only 236 other dogs (a rate per hundred of 52) and probably not very many others, for about one-half of the home dogs were under confinement (table IV).

TABLE IV—*Ravages of the stray dog compared with the home dog, unconfined and confined*

CLASSIFICATION	TOTAL	PERSONS BITTEN	RATE PER HUNDRED RABID DOGS	DOGS BITTEN	RATE PER HUNDRED RABID DOGS
Stray dogs	186	236	124	210	113
Home dogs unconfined	230	260	112	198	85
Home dogs confined	249	148	69	38	16

The same proportion holds good whether the total figures for the state are taken, or whether the figures for the smaller towns and rural communities, exclusive of Chicago, are tabulated

TABLE V—*Dogs bitten by rabid dogs in Illinois (exclusive of Chicago)*

CLASSIFICATION	TOTAL RABID DOGS	NUMBER BITING OTHER DOGS	PER CENT BITING OTHER DOGS	NUMBER OTHER DOGS BITTEN	RATE (PER HUNDRED RABID DOGS)
Stray dogs	69	24	34	226	301
Home dogs	163	36	22	118	115

(table V). In table IV the ravages of rabid dogs against other dogs are indicated. Not only do a greater proportion of stray dogs bite other dogs than do home dogs, but for each stray dog the rate is twice as high (and probably four or five times higher if all the figures were known) as is the rate for the home dog.

Next to the stray dog comes the unconfined home dog in the spread of rabies. While the rate is smaller than for the stray dog, it is much higher than the confined dog.

The control of rabies rests upon the destruction of stray dogs and the confinement of home dogs. Any program for the control of the disease which neglects these factors is bound to fail.

The role of the home dog: The total figures for the State would indicate that more people were bitten by stray dogs than by home dogs (table IV). This is especially true in the city of Chicago where the density of population makes any rabid dog upon the street very dangerous. A different result is obtained when the Chicago figures are separated from the rest of the State. In the smaller towns not only did a larger percentage of home dogs bite people that did stray dogs, but each home dog bit more people than did each stray dog (table VI).

TABLE VI—*Persons bitten by rabid dogs in Illinois (exclusive of Chicago)*

CLASSIFICATION	TOTAL RABID	NUMBER BITING PERSONS	PER CENT BITING PERSONS	PERSONS BITTEN	RATE (PER HUNDRED RABID DOGS)
Stray dogs	69	32	46	53	76
Home dogs	163	96	59	158	97

Emphasis is placed upon the fact, therefore, that, except in the largest cities, the home dog is more of a menace to people than the stray dog. For the protection of the family the home dog should be vaccinated against rabies.

SUMMARY

1. A study of the histories of 452 rabid dogs showed that 18 of them had received prophylactic treatment for rabies previous

to the onset of the disease. Ten of them developed symptoms within one month after vaccination and were apparently in the incubation stage of the disease when vaccinated. Three developed symptoms three and one-half months to six months after vaccination, which would possibly indicate a failure of protection by the vaccine.

2. The stray dog is the reservoir of infection in the perpetuation of rabies. It bites several times as many other dogs as does the home dog. The unconfined home dog comes next in order in biting other dogs, while the home dog that has been placed under the observation of a veterinarian at the first suspicious symptoms is an insignificant factor in the perpetuation of the disease. Any program for the control of rabies which neglects the confinement of dogs, with destruction of the stray animals, is bound to fail.

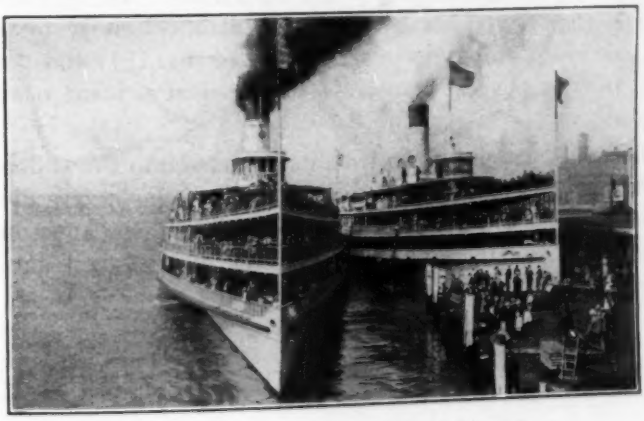
3. The home dog bites more people than does the stray dogs (except in the large cities, where the population is dense). For the protection of the family, therefore, the home dog should be vaccinated against rabies.

What a Relief

Ed. Lober, who has been assisting the veterinarians examining the chickens around here, states that they will be finished with their work in this township, Monday. After Monday, we can feel free to eat eggs.

Cissna Park (Ill.) *News*

Michigan is washed by all the Great Lakes but one.



Excursion Steamers on Detroit River

A DISSECTION OF THE NERVE SUPPLY TO THE GASTRIC COMPARTMENTS OF THE RUMINANT (BOVINE)

By H. L. Foust, *Ames, Iowa*

Division of Veterinary Medicine, Iowa State College

This dissection was attempted in order to supplement the somewhat meager descriptions found in available literature covering the nerve supply to the four divisions of the ox stomach. The knowledge of the arrangement of the nerves, both vagus and sympathetic, and their terminal distribution, was needed in a study of gastric physiology in the ruminant being conducted by my colleagues, Drs. A. F. Schalk and R. S. Amadon, in the Veterinary Department of the North Dakota Agricultural College.*

The massive collections of adipose tissue along the course of the nerves, even in apparently markedly emaciated individuals, have made the dissection of the nerves difficult and there should be further similar studies to verify and correct the findings which will be described, and which will be illustrated by drawings.

The work here presented is the culmination of the dissection of the cadavers of three cows.

The dorsal esophageal branch of the vagus nerves was formed dorsal to the esophagus at the level of the seventh thoracic vertebra by the junction of the dorsal esophageal branches of the right and left vagus nerves. It continued posteriorly and passed through the hiatus esophageus of the diaphragm, somewhat to the right of the dorsal part of the esophagus. An anastomotic branch (fig. 1, *3*) passing obliquely ventroposteriorly over the left side of the esophagus joined the dorsal (fig. 1, *1*) and ventral (fig. 1, *2*) esophageal branches of the vagi at a point near the hiatus.

Two to three centimeters from its entrance into the abdominal cavity, the dorsal esophageal branch of the vagus nerves divided into a number of branches. These with their anastomotic branches from the coeliaco-mesenteric ganglia will now be described

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*Acknowledgment is due to Dr. A. F. Schalk, Head of the Veterinary Department of the North Dakota Agricultural Experiment Station, Fargo, N. D., for his permission to make the dissections and for many helpful suggestions. The work was done while the writer was a member of the staff of the Department to which reference is made above.

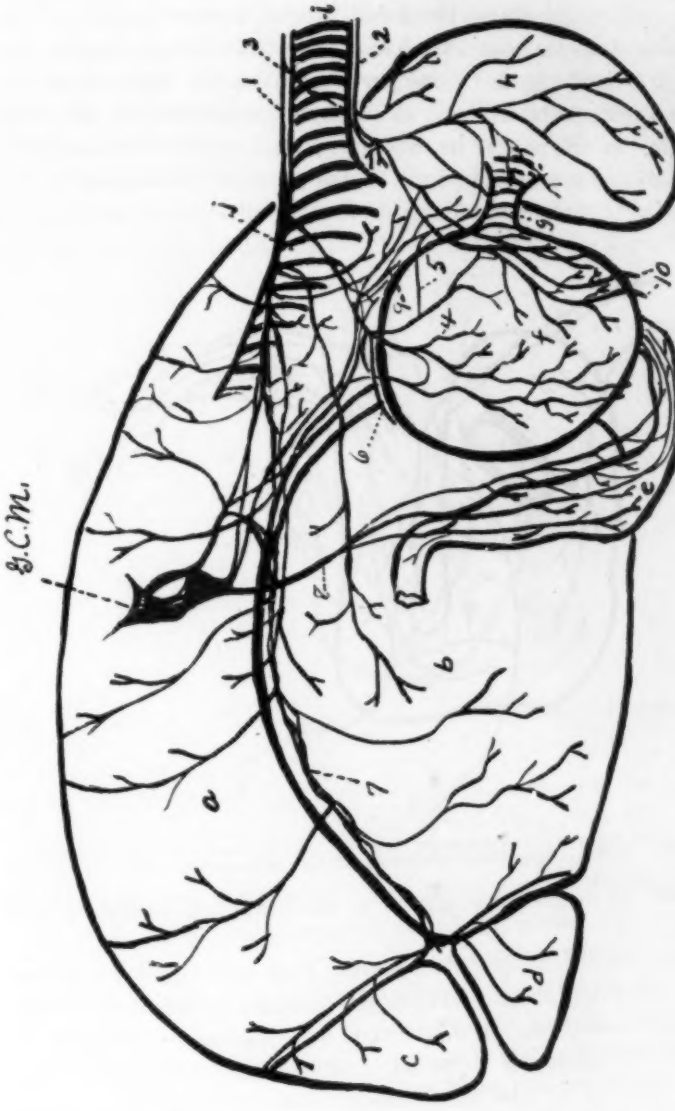

FIG. 1. Stomach of ox (right view). a, dorsal sac of rumen; b, ventral sac of rumen; c, d, posterior blind sacs of rumen; e, abomasum; f, omasum; g, reticulo-omasal connection; h, reticulum; i, œsophagus; j, atrium ventriculi; G.C.M., coeliaco-mesenteric ganglia.

1. After receiving anastomotic branches from the coeliaco-mesenteric ganglia, rami (fig. 1, *4*) with anastomosing branches formed a plexus along the course of the dorsal branch of the omasal-abomasal artery. Many small nerves were given off in its course which innervated the dorsal, lateral, posterior and ventral surfaces of the omasum. In close proximity to the abomasum the plexus divided (fig. 2, *1*), one part supplying the right while the other supplied the left side of the lesser curvature of the abomasum. In the part of its course proximal to the dorsal surface of the omasum, a branch (fig. 1, *5*) was given off which innervated a portion of the omasum dorsal to the reticulo-omasal connection

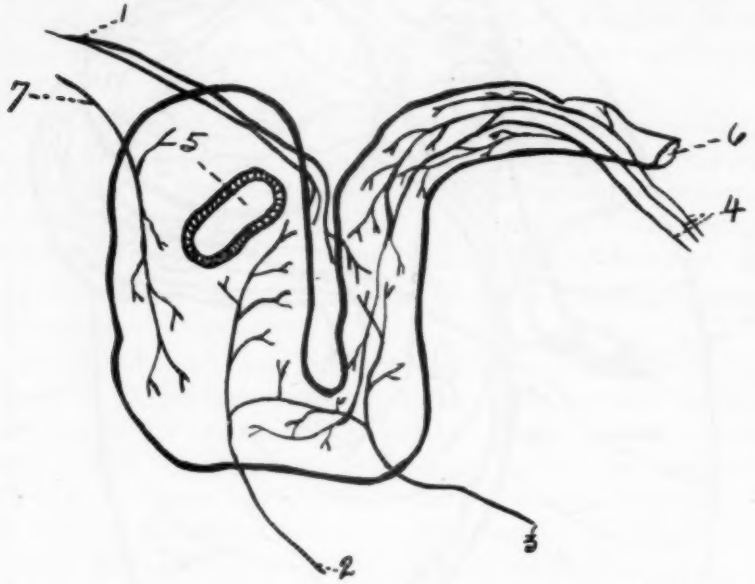

FIG. 2. Abomasum of ox (dorso-posterior view). 5, omaso-abomasal orifice; 6, duodenum. The pyloric portion is turned ventrally for a better perspective.

and sent an anastomotic branch to the branches described in 4 below.

From the plexus described above, branches (fig. 1, *6*) were given off which passed between the contiguous surfaces of the rumen and omasum. In their course over the omasum (fig. 3, *1*), nerves were given off to its left posterior and left sides. The plexus formed by the anastomoses between these two nerves divided near the abomasum; one group of nerves (fig. 2, *2*) supplied the left part of its dorsal surface near its lesser curvature, while the other group (fig. 2, *3*) went to the right part of its dorsal surface terminating on the pyloric part.

2. Several short branches supplied an area on the rumen immediately ventral and posterior to the atrium ventriculi.

3. Short nerves (fig. 1, *9*) were given off which supplied the dorso-medial surface of the omasum (fig. 3, *4*, *4'*, *4''*, *4'''*). Some sent branches to the reticulo-omasal connection.

4. Short rami to the reticulo-omasal tube. From one of these an anastomotic branch joined a plexus following the ventral branch of the omasal-abomasal artery. This latter plexus gave ramuli to the surface of the reticulum ventro-sinistral to the reticulo-omasal orifice and passed through the rumeno-reticular sulcus, innervating the postero-ventral surface of the reticulum and the anterior surface of the dorsal anterior blind sac of the

FIG. 3. Omasum of ox (left or medial view). 2, reticulo-omasal orifice; 3, omaso-abomasal orifice.

rumen and terminated by innervating the surfaces of the rumen and reticulum adjacent to the left end of the groove (fig. 4, *1*). A ramus was given off in the sulcus which innervated the left dorsal surface of the fundus region of the abomasum (fig. 2, *7*).

5. A plexus carrying branches both from the dorsal esophageal branch of the vagus and from the coeliaco-mesenteric ganglia followed the left ruminal artery through the anterior groove of the rumen. It gave ramuli to the dorsal and anterior surfaces of the ventral anterior blind sac of the rumen, also to the ventral and posterior sides of the dorsal blind sac of the rumen, and was continued at the left end of the groove by rami (fig. 4, *2*), some of which terminated by innervating the surfaces contiguous to the left part of the anterior groove while others following the left longitudinal groove and innervating the areas dorsal and ventral

FIG. 4. Stomach of ox (left view). a, dorsal sac of rumen; b, ventral sac of rumen; c, d, posterior blind sacs of rumen; e, abomasum; f, reticulum.

to it terminated by arborizing with the rami of the nerves from the posterior groove.

6. Rami containing both vagus and sympathetic fibers supplied the surface of the rumen, dorsal and to the left and posterior to the atrium ventriculi. Some passed over the dorsal surface terminating on the left side of the dorsal anterior sac. Others innervated the surface of the rumen ventral to and along the anterior part of the right longitudinal groove (fig. 1).

7. Rami continued posteriorly, receiving branches from the sympathetic plexuses near the origins of the branches of the coeliac artery and followed the right ruminal artery. From these rami (fig. 1, 7), which form a plexus, branches were given off to dorsal and ventral sides of the groove. Those running dorsally supplied the innervation for the right and dorsal surfaces of the dorsal sac of the rumen. Many of the dorsal branches passed over to the left face of the dorsal sac, terminating there. In one instance one branch passed over and beyond the left longitudinal groove and sent a branch posteriorly in the groove. The ventral ramuli innervated the right face of the ventral sac of the rumen.

At the coronary grooves, dorsal and ventral branches followed these grooves. These branches in turn gave off ramuli which terminated in the adjacent surfaces of the dorsal and ventral sacs and the dorsal and ventral posterior blind sacs of the rumen. The dorsal branch terminated on the dorso-lateral surface of the dorsal posterior blind sac. The ventral branch followed the ventral coronary groove terminating ventrally at about its middle.

The main plexus followed the right ruminal artery through the posterior groove of the rumen. It gave off branches to the dorsal and ventral posterior blind sacs and terminated by dividing into three rami which followed the left dorsal and ventral coronary and left longitudinal grooves (fig. 4, 3). The dorsal branch terminated a short distance beyond the end of the groove, the ventral continued in the ventral groove to about its middle, where it ended; and the anterior branch passed forward in the left longitudinal groove to terminate a little posterior to its middle. All three of these rami gave off collateral branches which innervated the surfaces of the rumen on both sides of their respective grooves.

The ventral esophageal branch of the right and left vagus nerves was formed about 6 cm. anterior to the point at which the dorsal branches joined. It ran posteriorly ventral to the esophagus, receiving an oblique branch from the dorsal nerve,

passed through the hiatus esophageus and bent ventrally at a right angle (fig. 1, *2*). It divided as follows:

1. Branches passed to the lateral, anterior and medial surfaces of the reticulum (fig. 1).

2. Short branches went to the anterior part of the atrium ventriculi (fig. 1).

3. Ramuli receiving anastomoses from the coeliaco-mesenteric-dorsal-vagus plexus innervated the dorsal and anterior surfaces of the reticulo-omasal connection (fig. 1).

4. Branches terminating in the left and ventral sides of the omasal-abomasal connection.

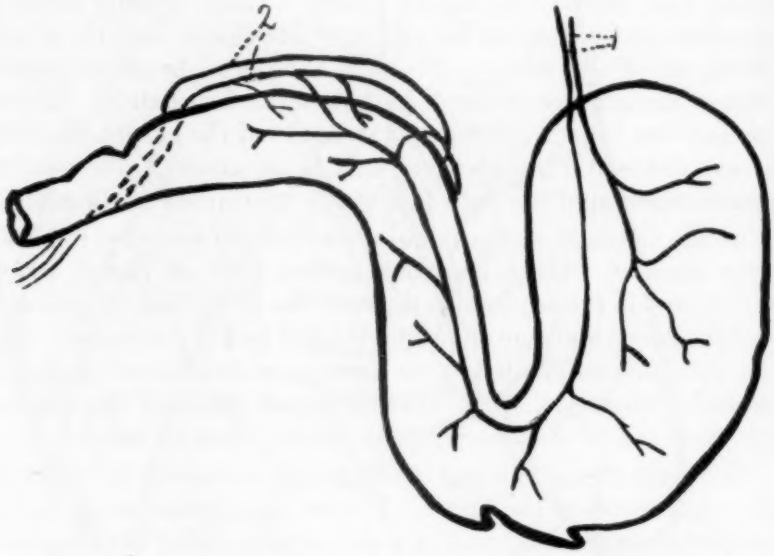

FIG 5. Abomasum of ox (ventro-anterior view). The pyloric portion is turned ventrally for a better perspective.

5. Branches, in most cases two (fig. 1, *10*), passed over the right anterior surface of the omasum, innervating this surface and terminating in the abomasum. Rami went to the right and left sides of the ventral surface of this organ on which their terminal ramuli were distributed (fig. 5, *1*).

The coeliac and anterior mesenteric ganglia of each side were connected to each other by bands running transversely both anterior and posterior to the root of the coeliac and anterior mesenteric arteries. From these ganglia, plexuses followed the branches of the arteries toward their terminations. In their

course they gave off anastomosing branches to both the dorsal and ventral esophageal branches as well as supplying branches to the viscera over which they coursed. A remarkably large branch followed the gastro-duodenal artery to the pyloric part of the abomasum. Its distribution is illustrated in fig. 1, *8*, fig. 2, *4* and fig. 5, *2*.

Oklahoma Meeting

Dr. C. H. Fauks, secretary of the Oklahoma State Veterinary Medical Association, announces that the mid-summer meeting will be held at Bartlesville, Okla., July 9-10. A very interesting program is being prepared. It will include a visit to Mr. Frank Phillips' beautiful wild animal ranch on the first day of the meeting. Veterinarians from other states are especially invited to attend.

Book Building and Tower, Detroit

CAPILLARIA IN CHICKENS

By ROBERT GRAHAM, FRANK THORP, JR. *and* R. L. HECTORNE

Laboratory of Animal Pathology and Hygiene, University of Illinois, Urbana, Illinois

During the month of January, 1929, chickens from two different and widely separated Illinois farm flocks, upon postmortem examination, proved to be infested with the nematode, Capillaria. The species may be Annulata, though the confusion in literature on Capillaria species makes an accurate identification of the parasite somewhat doubtful. The parasites from case 1 were referred to nematologists. The pathologic changes encountered

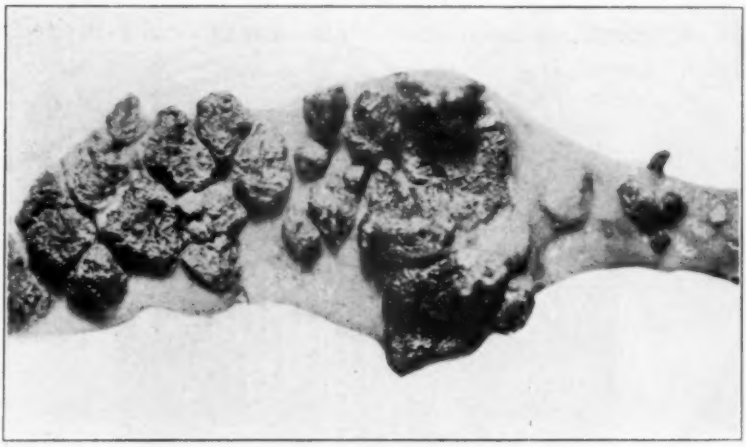

FIG. 1. Follicular diphtheritic enteritis.

in the ingluvium and esophagus in affected fowls of one flock resemble the gross lesions ascribed to *Capillaria annulata* infestatons. The difference in the gross lesions at autopsy in specimens examined suggest the possibility of different species of capillarids being involved.

Cram[1,2] reported finding *Capillaria annulata* in affected grouse, in Michigan; in chickens, from Georgia, Mississippi and Louisiana; and from turkeys, in Maryland. Jungherr[3] reported the same parasite in turkeys in Montana. Hung[4] described the histopathology of *Capillaria annulata*, which resembles lesions encountered in fowls in case 2 coming to our attention. It is possible that the character and extent of the intestinal capillariasis

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may ultimately aid the autopsist in distinguishing different species of this parasite.

*Case 1:** In a group of 100 chickens, that were purchased from a local poultry dealer, one sickly fowl came to our attention. The affected fowl showed symptoms of weakness, anemia, and emaciation over a period of several days. At autopsy the intestine just anterior to the ceca showed a marked dilatation. The intestinal contents had a very putrid odor while a follicular, diphtheritic enteritis of the inner wall was found in the dilated intestine (fig. 1). Histopathologic study of the intestinal lesions

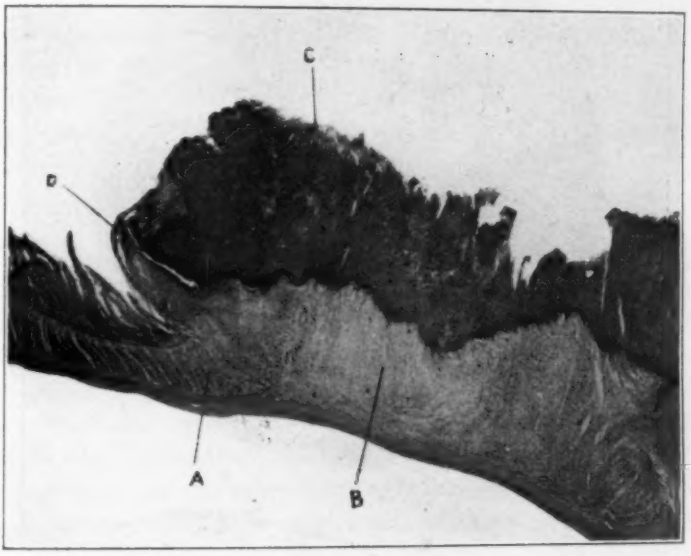

FIG. 2. Follicular diphtheritic enteritis accompanied by necrosis of the epithelium and fibrosis of the submucosa and muscular wall. A, junction of circular muscle layer and invading fibrous tissue. B, fibrosis of submucosa. C, necrosis of the epithelium. D, junction of fibrotic submucosa and area of cellular infiltration.

revealed necrosis and sloughing of the intestinal epithelium. The submucosa and muscularis showed infiltrating areas of fibrin filaments, and a chronic fibrosis (fig. 2). The disintegration and replacement of the circular muscle fibers explain the intestinal dilatation. Scrapings of the necrotic membrane upon microcsopic examination revealed masses of double operculated eggs (figs. 3 and 4) and a small number of capillarids. The

*Doctor H. B. Ward, Professor of Zoology, University of Illinois, independently identified the genus as Capillaria in case 1. Since the above report was prepared, Doctor Maurice C. Hall, Chief of the Zoological Division, U. S. Bureau of Animal Industry, Washington, D. C., advised that Miss Cram regarded the species referred to in case 1 as *Capillaria meleagris-gallapavo*.

mature parasites, though minute, could be seen with the unaided eye, when washed free of débris on glass slides.

Case 2: In another farm flock of 300 chickens, a parasite of the same genus was found in several sick chickens. The symptoms consisted of emaciation, lack of vigor, droopiness, lameness, ruffled feathers, torticollis and, finally, extreme helplessness and death. Affected fowls died in ten days to five weeks after dis-

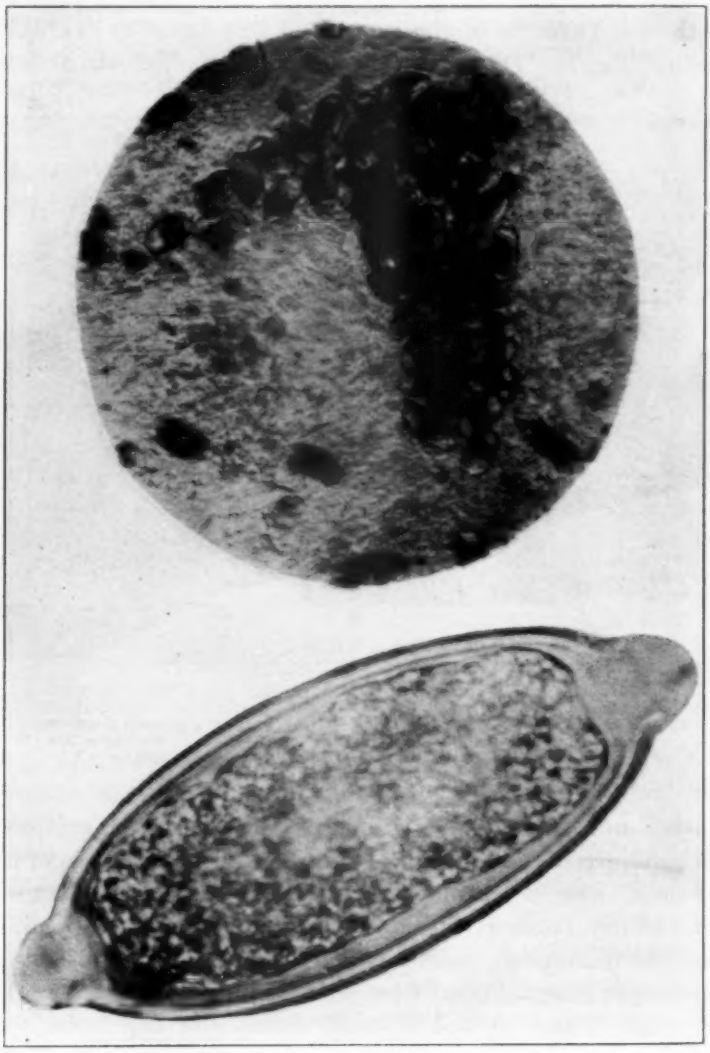

FIG. 3. (Above) Eggs of capillaria in the diphtheritic membrane of the intestines shown in figure 1. (Low magnification.)

FIG. 4. (Below) Egg of capillaria. (Highly magnified.)

playing symptoms. The losses continued throughout the fall and early winter months. The late hatches were most affected. Pullets weighing 2½ to 3 pounds were first affected. At the time specimens were submitted for examination, a total of 35 chickens had died.

The gross pathology in affected fowls were confined to the lower portion of the esophagus and ingluvium, as described in *Capillaria abbulata* infestations. A putrid odor was noted on opening these organs. Careful examination revealed an ingluvitis. The lymph-follicles of the wall of the ingluvium were enlarged and a slightly adherent diffuse fibrinous pseudomembrane extended over the surface. Small circumscribed patches of the mucosa were necrotic and easily displaced on gentle pressure. Scrapings from the inflamed ingluvium revealed a few capillarids embedded in the fibrinous membrane and necrotic epithelium, with many double operculated eggs. The eggs were indistinguishable from those encountered in case 1 (see figures 3 and 4).

Summary

The symptoms and pathologic lesions encountered in chickens from two different flocks in Illinois were, as far as could be determined, traceable to the presence of the nematode Capillaria. The possibility of two separate species of this parasite in cases 1 and 2 is recognized. These parasites impair the function of the ingluvium and intestine. The gross lesions in one specimen were confined to the intestine, while the ingluvia of several fowls examined from the other flocks were involved. It is possible that capillarids are more prevalent than supposed, yet in conducting several hundred autopsies, the above cases represent the first time capillarids, to our knowledge, have been recognized as a pathogenic agent in farm flocks in Illinois. Necropsy findings, fortified by microscopic and histopathologic examinations, may enable veterinarians to recognize parasitic ingluvitis or enteritis in fowls from enteric inflammations due to other causes.

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[4]Hung, See Lü: Pathologic lesions caused by *Capillaria annulata*. No. Amer. Vet., vii (1927), 10, pp 49-50.

The ideal convention city of the summer months—Detroit, Michigan—welcomes the A. V. M. A., August 13-16.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

A CASE OF TUBERCULOSIS IN A COWBIRD*

By C. B. HUDSON *and* F. R. BEAUDETTE

New Jersey Agricultural Experiment Station, New Brunswick, N. J.

The case of tuberculosis to be recorded here occurred in a cowbird (*Molophrus ater*) which was sent to us through the courtesy of Mr. B. S. Bowdish, of the New Jersey Audubon Society. The following history accompanied the specimen:

> I am sending . . . a cowbird which was found in an apparently injured condition in a stable of a neighbor of mine several days ago. The breast protruded on the left side, and the bird was unable to fly with any strength or for any distance. We kept it over night and provided food and water, and the next day it seemed so much better that it was released, only to be returned to us that evening by the same neighbor, having been found again in the immediate vicinity of his stable. It lived for several days though gradually getting weaker and feeding less, and during the last twenty-four hours, was not observed to eat at all. Unfortunately it died on Saturday afternoon.

The specimen was kept on ice until the following Tuesday, at which time it was sent. By the time the bird reached us, considerable decomposition had taken place. Autopsy revealed generalized tuberculosis which was confirmed by the finding of myriads of acid-fast organisms in the nodules of the liver.

The literature on avian tuberculosis is voluminous, but deals mostly with the disease in the common fowl. Not a few cases are reported in turkeys, but ducks, geese, guineas and pea fowls appear to be rarely affected. Jones[1] reported upon an outbreak of tuberculosis in pigeons, and Riddle[2] gave an excellent account of the distribution of lesions in 940 Columbidae. Tuberculosis of parrots has been reported by several investigators. Eberlein[3] records the occurrence of 56 cases in 154 parrots treated at the Berlin clinic. He, like many others, considers the human type of organism to be involved in parrot tuberculosis, and argues that infection from other sources would be difficult to explain. Hobday[4] observed a growth, at the entrance of the thorax of an ostrich, which was removed by him. Stained preparations from

*Journal Series Paper of the New Jersey Agricultural Experiment Station, Department of Poultry Husbandry. Received for publication, January 11, 1929.

the excised growth revealed tubercle bacilli. The ostrich (Nancy Hanks) had been reared in California and was sent to England, where it came to Hobday's attention. Later the bird died at sea, while in transit to Antwerp.

Koch and Rabinowitsch[5] examined the birds dying in the Berlin garden from December, 1903, to August, 1905, and found tuberculosis in 118 out of 459 specimens. In all, fifteen orders were represented and representatives of ten of them had the disease. The few orders in which the disease was not found were represented by only a few specimens.

Canary birds are said to be affected with tuberculosis, but the existence of a psuedo-tuberculosis, which is much more common, causes some doubt to be raised regarding the prevalence of tuberculosis in this bird.

Tuberculosis of pheasants has been reported by Morse, and we have also found the disease in birds sent to us from a New England state. The pheasants had been reared in captivity.

Thus, from the brief review, it will be observed that most of the tuberculosis reported in birds has been in domesticated species, or, in wild birds reared or kept in captivity under conditions which would enable infection to be contracted from another source. The occurrence of the disease in wild birds, as such, has been observed by few, at least the literature leaves that impression. Metchnikoff, however, seems to have found the disease in a sparrow, and Van Es and Schalk[6] observed a well-marked case of tuberculosis in a sparrow which had access to an infected poultry-yard. These investigators demonstrated the infectiousness of chicken tuberculosis for sparrows by producing fatal generalized tuberculosis in twelve of them by a single feeding of tuberculous chicken liver.

With respect to tuberculosis in wild birds, Van Es and Schalk say: "We know little of its existence among free wild birds. It is probable that it occurs among such scavengers as the common sparrow."

Further inquiry developed the fact that this bird (a male, No. 580812), with its mate, had been taken on two previous occasions in the trap of Mr. Bowdish, at Demarest, Bergen County, New Jersey. This is evidence that the bird was a local one. The owner of the stable in which the bird was found, does not keep chickens nor have any been on the premises for years. The question as to where the disease was contracted would be difficult to answer, in view of the fact that tuberculosis of poultry is of

rare occurence in New Jersey. In fact, we have never diagnosed the disease in poultry from Bergen County, and the only cases coming to our attention from the examination of 13,306 specimens since 1923 are listed below:

Date	*Town*	*County*
November 6, 1923	Pennington	Mercer
November 12, 1924	Frenchtown	Hunterdon
March 10, 1924	Old Bridge	Middlesex
April 1, 1924	Brought in by Extension Specialist	
April 1, 1924	Succasunna	Morris
October 8, 1924	Blawenburg	Somerset
December 15, 1924	New York City*	
December 16, 1924	New Brunswick	Middlesex
February 2, 1925	Somers Point	Atlantic
March 31, 1925	Belvidere	Warren
April 7, 1925	New Brunswick	Middlesex
December 19, 1925	Hightstown	Mercer
June 9, 1926	Sand Brook	Hunterdon
January 10, 1927	Glen Moore	Mercer
January 21, 1927	Chatham	Morris
March 1, 1927	Belvidere	Warren
August 8, 1927	Princeton	Mercer
November 17, 1927	Essex Fells	Essex
December 8, 1927	Pennington	Mercer
September 5, 1928	Clover Hill	Hunterdon
September 20, 1928	Mt. Holly	Burlington
October 16, 1928	New Providence	Union

*Died of fowl plague.

The injury referred to in the history reminds one of the numerous injuries found in grouse during an investigation of the so-called "grouse disease." This "epidemic" of injuries, resulting from collision with objects, was thought to be due to impairment of flight as a result of the "grouse disease." Impaired flight had been observed in the cowbird and it is quite likely that the injury was acquired in this manner.

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In the year of our Lord, 1929, and of the A. V. M. A., 66, you can combine business and a pleasurable vacation. Detroit, August 13-16.

OPERATIVE RELIEF FOR OVA IN THE PERITONEAL CAVITY OF THE CHICKEN

By FRANK D. MCKENNEY, *Rochester, Minnesota*
Division of Experimental Surgery and Pathology, The Mayo Foundation

The condition of loose eggs in the abdominal cavity is seen quite frequently in hens. The condition is associated with heavy production. It has been suggested that infection of the ovary and congestion of the oviduct cause reverse peristalsis of the latter,

FIG. 1. The hen before operation, showing characteristic posture assumed.

and that this pushes the egg into the abdominal cavity instead of allowing it to descend to the cloaca. The condition may also be caused by a ruptured oviduct due to obstruction. In this case the possibility of restoring the usefulness of the oviduct is rather remote, but the fowl can still be used for show purposes or can be dressed for market.

The causes of this condition have been briefly described by Ward and Gallagher,[1] but as far as could be ascertained from the literature, nothing has been reported on the treatment or the

Received for publication April 3, 1929.

possibility of an operation to restore the function of the ovary and the usefulness of the hen. The value of the average hen would not warrant such an operation but in the pedigreed stock of the poultry fancier an operation would be of value because the mortality from this condition is very high. Death usually is due to peritonitis.

The case which came under my observation was that of a pullet from a pedigreed flock which was on a program of production. The pullet affected with this condition had the history of laying

FIG. 2. The hen eight days after operation.

very large or double-yolked eggs. Clinical signs developed quickly and assumed an attitude characteristic of this condition. Instead of the body being held in a horizontal plane, the posture of a duck was assumed, with the posterior portion of the sternum and abdomen very close to the ground (fig. 1). The feathers were worn off below the vent, because of contact with the ground.

The condition was diagnosed as due to a loose egg in the abdominal cavity and an operation was performed. The field was cleaned by plucking the feathers below the vent and painting the

area with iodin. Ether anesthesia was used, and ordinary aseptic measures were employed. An incision 7.5 cm. in length was made between the xiphoid portion of the sternum and the cloaca, and the eggs were removed. Four whole eggs, three of which had soft shells, and parts of two broken eggs were found. All the eggs were very large. The large egg-yolks found in the egg cluster were then compressed, forcing them out of the ovary to prevent more eggs from forming before the parts had become normal. The incision was closed with a continuous suture of catgut. The pullet has made an uneventful recovery and is in excellent condition. She laid the first egg forty-six days after operation (fig. 2).

This operation affords a means of procedure for the practitioner interested in poultry practice, and since the demand for fancy breeding stock is steadily causing increases in the price paid for poultry of this kind, this operation is now worth considering. Fowls become anesthetized under administration of ether so easily that care must be taken that they do not expire, but they are rather resistant to peritonitis. Special instruments or technic are not essential, and only a few minutes are required to perform the operation.

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Drive to Dynamic Detroit.

RICKETS AS A SECONDARY MANIFESTATION IN COCCIDIOSIS

By H. J. Stafseth, *East Lansing*

Department of Bacteriology Michigan State College

The addition of cod-liver oil and lime to the ration of growing chicks has been advocated mainly for the purpose of preventing rickets. It is claimed also that supplementing feed with these ingredients helps to produce better chicks in general. Most up-to-date poultrymen have had a chance to test the validity of these claims and invariably feel that they are sound. So much greater, then, is their surprise when they find that their chicks develop typical symptoms of rickets while being fed a perfectly balanced ration supplemented with cod-liver oil, lime and greens.

For several seasons we have had the opportunity to examine rachitic chicks that had been fed cod-liver oil and lime, more or

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less continuously. Some of these chicks came from poultry establishments where everything possible is done to promote health.

It has invariably been our experience to find that these chicks show extensive enteritis, usually due to coccidiosis, and that the feeding of powdered milk, as recommended by Dr. J. R. Beach, will eliminate this trouble.

It seems perfectly possible that microorganisms other than coccidia or intestinal parasites, capable of causing extensive and lasting enteritis, might bring about similar results.

The most logical explanation that can be offered at this time, for an apparent connection between coccidiosis, or enteritis brought about by other causes, is that the extensive injury of the intestinal mucous membrane interferes with proper digestion and assimilation.

While this problem has not been studied experimentally, I feel that we have sufficient proof for the existence of this condition and since practicing veterinarians and others engaged in the control or study of poultry diseases may encounter similar circumstances, I have thought it worthwhile to call attention to our observations on this subject.

Meet me in Detroit, August 13-16, where life is worth living.

A CASE OF WHEAT-BEARD INJURY IN PIGS

By L. P. Doyle, *Lafayette, Ind.*

Department of Veterinary Science

Purdue University Agricultural Experiment Station

Three 3-weeks old pigs were received for examination. The history of the trouble indicated that a large proportion of the spring pig crop on this particular farm had died before the pigs were three weeks of age.

Autopsy findings: All of the pigs showed raised, long, narrow, yellowish-white, apparently necrotic areas in the walls of the gastrointestinal tract or under the peritoneum. One pig showed such areas on the abdominal surface of the diaphragm, on the surface of the spleen, and several in the wall of the stomach. The other two pigs each showed several of these areas in the wall of the

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colon. One also had a similar area in and under the parietal peritoneum in the region of one kidney.

The apparently necrotic areas were found to contain long, sliver-like pieces of woody material which were identified by Dr. E. B. Mains, of the Botany Department, as wheat beards. The beards were well preserved, although they were completely encapsulated in the pigs' tissues.

It does not seem likely that the wheat beards were solely responsible for the heavy losses in the pigs. It is probable that the pigs were not normal when they swallowed the beards. However, the case shows that wheat beards are able to penetrate the walls of the gastrointestinal tract of young pigs, and migrate into other organs.

Motor to the motor metropolis, August 13-16.

A GENERALIZED MELANOSARCOMA IN A MULE

By FRANK P. MATHEWS, *Lafayette, Ind.*

Department of Veterinary Science,

Purdue University Agricultural Experiment Station

The subject in this case was a mule, seventeen years of age. The animal had been losing weight, and had refused food and

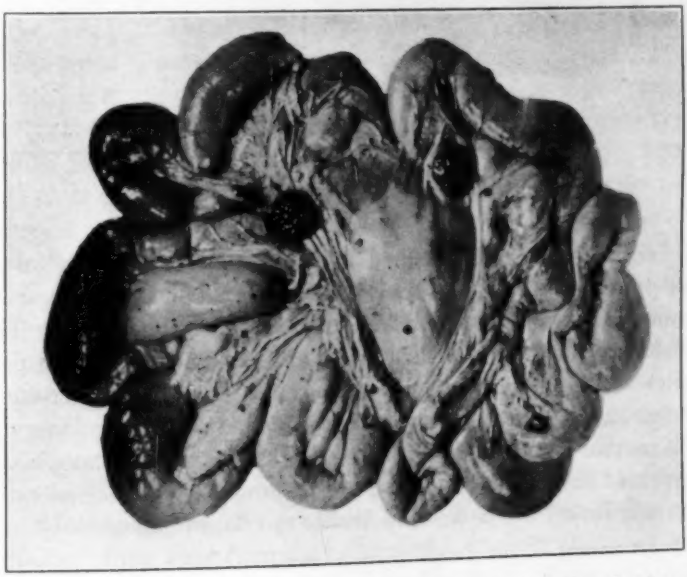

FIG. 1. The intestine showing numerous melanomas attached to the peritoneal surface.

water for several days before it was presented to the laboratory for autopsy.

The external lesions consisted of a firm swelling on the right side of the brisket, which extended anteriorly up the neck for about a foot and laterally to about the point of the shoulder. The owner claimed that this swelling was of eleven months duration. Upon autopsy several subcutaneous melanomas were found. The swelling in the region of the sternum proved to be a black, diffused growth, which extended into the throracic cavity and involved the anterior mediastinal lymph-glands. Pressure upon the esophagus by an enlarged esophageal lymph-gland had evidently interfered with deglutition. The lungs contained several melanotic tumors, ranging from a few millimeters to two or three inches

FIG. 2. The spleen showing three metastatic tumors.

in diameter. The liver weighed 35 pounds and was the seat of an undetermined number of melanomas. The spleen weighed 17 pounds and showed an extensive neoplastic involvement. The mesentery was dotted with a large number of pigmented tumors which stood out in bold relief against the glistening, peritoneal background. Both kidneys were involved, and a tumor the size of a marble was found in the meninges. As a rule the neoplasms presented the typical black color of melanomas, but occasionally one was found which was practically devoid of pigment. Upon incision some of the larger tumors also revealed gray, non-pigmented areas.

A microscopic examination of sections taken from the pigmented portions showed the typical, melanin-laden, oval to round cell, with a single nucleus crowded towards the periphery. In many fields this was found to be the predominating type of cell. The non-pigmented areas presented a tissue composed of large polyhedral cells, with poorly-defined cell boundaries. Each cell contained a round, vesicular nucleus. Melanin was demonstrated in some of these cells, but it was not unusual to find entire fields composed of non-pigmented cells. In such fields mitotic figures were frequently observed.

The evidence of rapid growth and wide dissemination in this case is analogous to the early metastasizing, rapidly fatal melanosarcoma of man, a syndrome not commonly observed in melanomas of the lower animals.

If you remember the last meeting of the A. V. M. A. in Detroit, you will not be found absent from the Dynamic City, August 13-16.

FOREIGN BODIES IN THROAT OF COW

By E. D. WINELAND, *Booneville, Ark.*

On April 10, I was called to see a cow five miles southwest of Booneville. Upon arrival, I found the cow suffering with difficult breathing and a cough. I suspected pneumonia, but, after taking the temperature, I eliminated that disease. I inserted my mouth speculum and examined the throat. I found and removed a piece of cob two inches long. Thinking that I had removed the cause, I returned home. The second day I received another call and found the condition worse. I proceeded to make another examination and this time I removed a piece of baling wire four inches long from the upper part of the larynx. The cow made a nice recovery and is still doing fine. The cow belonged to Mrs. Ida Oliver, of Booneville. I submit this report, as other veterinarians may have similar cases and may profit by my experience.

Michigan has the greatest coast-line of any state. The line forms at the right, in Detroit, August 13-16, led by the A. V. M. A.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Captain Louis L. Shook is relieved from duty as assistant attending veterinarian, Washington, D. C., and assigned to duty at West Point, New York, effective on or about August 20, 1929.

Lt. Colonel Robert J. Foster is assigned to duty at Omaha, Nebraska, effective upon completion of his present tour of foreign service in the Panama Canal Department.

Captain Chauncey E. Cook is assigned to duty at Fort Leavenworth, Ks., effective upon completion of his present tour of foreign service in the Panama Canal Department.

The appointment of Second Lieutenant Arvo Theodore Thompson, Veterinary Corps Reserve, as second lieutenant in the Veterinary Corps, Regular Army, with rank from April 8, 1929, is announced. Lieutenant Thompson will report to the commandant, Army Medical School, Army Medical Center, for duty.

Each of the following-named officers of the Veterinary Corps is relieved from his present assignment and duty at the station specified, at such time as will enable him to comply with orders, and will proceed to Washington, D. C., and report to the commandant, Army Veterinary School, on or about August 29, 1929, for the purpose of pursuing a course of instruction:

Captain George W. Brower, Fort D. A. Russell, Wyoming.
Captain John R. Ludwigs, Fort Monroe, Virginia.
Captain Gardiner B. Jones, Fort McIntosh, Texas.
Captain Arthur D. Martin, Chicago Quartermaster Depot, Chicago, Ill.
Captain Frank M. Lee, Fort Jay, New York.
Captain Gerald W. FitzGerald, Kansas State Agricultural College, Manhattan, Ks.

Orders on Captain Philip H. Riedel have been amended so as to direct him to report for duty at Fort Jay, New York, upon completion of his present course of instruction at the Medical Field Service School, Carlisle Barracks, Pa.

Reserve Corps

Promotions

Cunningham, Geo. Nelson to . Captain . . . 1201 W. Harrison St., Freeport, Ill.
Conrad, Burton Wesley to . . . Captain . . . 928 Main Street, Sabetha, Kansas.

New Acceptances

Moulthrop, Ralph Roy . . . . . 1st Lt. . . 45 Oakwood Avenue, Lawrence, Mass.

North Carolina Meeting

The dates for the summer meeting of the North Carolina State Veterinary Medical Association, as announced in the May issue of the JOURNAL, were in error. The correct dates are June 26-27-28. Secretary Scarborough announces that he had secured some notable talent for the program, including Dr. Adolph Eichhorn, Director, Veterinary Department, Lederle Antitoxin Laboratories; Dr. H. J. Milks, New York State Veterinary College, Cornell University; Dr. W. E. Cotton, Superintendent, B. A. I. Experiment Station, Bethesda, Md.; and Dr. C. E. Salsbery, Jensen-Salsbery Laboratories, Kansas City, Mo.

COMMENCEMENT

ONTARIO VETERINARY COLLEGE

The spring examinations of the Ontario Veterinary College, Guelph, Ontario, were completed April 23, 1929, and graduation exercises were held on April 30.

The degree of Bachelor of Veterinary Science was conferred at a special Convocation of the University of Toronto on eighteen graduates as follows: Thomas P. Bett, L. N. Brown, E. E. Carlson, G. H. Collacutt, T. J. Cornish, Norman H. Cox, J. R. Currey, S. A. Davidson, I. Erickson, Thomas N. Fortune, William H. Gear, Sydney H. Holloway, Arnold H. Kennedy, Hugh E. Macdonald, Robert A. McIntosh, Thomas Moore, Edward K. Seale, Alan C. Secord.

Honors were awarded to members of the graduating class as follows:

General Proficiency

First Prize—S. H. Holloway, of Dowling, Alta.
Second Prize—E. K. Seale, of Salem, Mass.
Third Prize—C. H. Collacutt, of Port Perry, Ont.

Bacteriology

Special Prize—S. H. Holloway, of Dowling, Alta.

Helen Duncan McGilvray Honorarium

E. E. Carlson, of Milestone, Sask.

Drive your car, family-full, back to its birth-place—Detroit, Michigan—August 13-16

K. S. A. C. PRIZES

The sixth annual Recognition Day program of the Kansas State Agricultural College took place in the College Auditorium, May 3, 1929. At this time, all winners of student prizes in the College received their prizes and awards. The names of the winners of the prizes offered the students of the Division of Veterinary Medicine are as follows:

General Proficiency

(Prizes offered by Dr. E. A. Schmoker (K. S. A. C. '17), of Everett, Wash.)

First Prize ($10.00)..... Karl Willim Niemann
Second Prize ($5.00)..... Lawrence O. Mott

Therapeutics

(Prizes offered by Jensen-Salsbery Laboratories, of Kansas City)

First Prize ($7.50)..... Wesley Watson Bertz
Second Prize ($7.50)..... Henry Devore Smiley

Pathology
(Prize of $7.50 offered by the Veterinary Faculty of the College)
Karl Willim Niemann

Physiology
(Prize of $7.50 offered by the Veterinary Faculty of the College)
Andrew Lafayette McBride

In addition to the prizes in the Division of Veterinary Medicine, two of the senior students, Karl William Niemann and Lawrence O. Mott, have been elected to membership in the Gamma Sigma Delta, the honor society in agriculture, and one of the junior students, T. J. Muxlow, has been elected to the honor society in military science.

The A. V. M. A. meeting in Detroit August 13-16, will be truly international—Canadian-American

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Minnesota Legislature Adopts Resolution

The following joint resolution was adopted by the Minnesota Legislature at the forty-ninth session:

WHEREAS, There is an ever-present danger of foot-and-mouth disease and other dangerous and fatal contagious diseases being imported into the United States, through the importation of live stock, meat and animal products from countries where such diseases exist, and

WHEREAS, The presence in this country of such dangerous and fatal contagious diseases would be disastrous to the live stock and dairy industries of the United States and to the health and welfare of its citizens, and

WHEREAS, It is a well-known economic fact that the prosperity of our country is dependent upon the prosperity of the live stock and agricultural industries in the United States, and

WHEREAS, The steps and measures heretofore taken by the Secretary of the United States Department of Agriculture and by other federal authorities for the prohibition and prevention of the entry into the United States of live stock, meat and animal products from countries where the foot-and-mouth and other dangerous and fatal contagious diseases exist meet with the hearty approval and endorsement of this Legislature and the people of this State, therefore, be it

Resolved, By the Senate of the State of Minnesota, the House of Representatives concurring, that the President of the United States, the Secretary of Agriculture of the United States, the Chief of the United States Bureau of Animal Industry and the Congress of the United States hereby are urgently petitioned and requested to continue the prohibition and prevention of the entry into the United States of live stock, meat and animal products from countries where the foot-and-mouth and other dangerous and fatal contagious diseases exist, and to take such further and other steps and measures as will fully and completely protect the live stock and agricultural industries of the United States from the dangers incident to the entry into this country of such diseases, and be it further

Resolved, That copies of this resolution be forwarded to the President of the United States, the Secretary of Agriculture of the United States, the Chief of the United States Bureau of Animal Industry and the members of Congress.

MISCELLANEOUS

To the Auxiliaries

When "we auxiliaries" entered the veterinary profession, we entered not via a college course and a D. V. M. degree as did our husbands, but through matrimony and a "Mrs." As we learned to comprehend the queer terminology, we gained such a knowledge of the veterinary ideals and hopes, that we soon made it our profession in a very real sense.

To be sure, we did and do not go out to vaccinate pigs, pull a horse's teeth, nor yet to practice quackery, even to the extent of telling a frantic neighbor what to do for her pet dog with distemper. Not these technical duties, but others more feminine, we are individually attempting with considerable earnestness. Collectively, we work together in our national auxiliary and in our state and district groups.

As we have read the JOURNAL and other veterinary publications, and have anticipated and made our annual pilgrimages to Lexington, Philadelphia and Minneapolis, we have learned about various troublesome problems, but few which have touched our own professional obligations as has the ever-present problem of publicity. This touches us in an unescapable way, being professional, but not technical. We have all laughed at the very, very. old story of telegraph, telephone and tell-a-woman, yet we know in our hearts that we really are good publicity agents.

Our profession needs to be better understood by the American public. This has to do not merely with what is written in our own journals, nor even in the magazines and papers of a general nature, but with the social and community esteem of our husbands and the whole profession. Almost everything that we do and say helps or hinders. Even our attitudes in little things, such as the social atmosphere that we create in our homes and in our church and club circles, may help so materially.

In various community circles, there are thousands of opportunities for this indirect education of the public, and sometimes for more direct education. For instance, a woman with a flare for organization, may well direct her energies to better milk production and distribution for her community. Another, with clubs as her hobby, can see that their programs have good lectures on

meat and milk and food inspection or, occasionally, every woman is asked a direct question. Then she may enlighten her hearers as to the scope and extent of the veterinary field. They will be surprised; for little do they know of our profession unless we tell them.

I submit this as being worth thinking about and considering informally next summer, when we meet again for a joyous good time in Detroit.

H. H. B.

Detroit is the home of pharmaceuticals.

Veterinarians Purchase Arabian Stallion

Three Sioux City veterinarians are active members of the nationally known Abu-Bekr Mounted Patrol which will participate in the Shrine ceremonials in Los Angeles during the first week in June. Dr. G. P. Statter is First Lieutenant and Veterinarian of the Patrol. Drs. D. D. Tobias and F. W. Cairy, the two other veterinary members, recently purchased the beautiful white pure-bred Arabian stallion, "Al Bedu," and added him to the other famous mounts belonging to the Patrol. "Al Bedu" is a direct lineal descendent of the famous stallion, "Skrowronek," now owned in England and recently featured in an article in the *Literary Digest*. "Abu Bekr" is another famous stallion belonging to the Mounted Patrol group and is ridden by Dr. Statter on frequent occasions. The Patrol will take about thirty mounts to Los Angeles.

Massachusetts Conference and Clinic

The Massachusetts Veterinary Association will hold a veterinary conference and clinic at the Eastern States Exposition grounds, Springfield, Mass., on Thursday, June 20, following the Eastern States Tuberculosis Eradication Conference, on June 18 and 19. Secretary Jakeman announces an attractive program. The veterinarians from outside the State who will contribute include: Dr. Adolph Eichhorn, Director, Veterinary Department, Lederle Antitoxin Laboratories; Dr. William J. Lentz, Director, Small Animal Clinic, University of Pennsylvania; Dr. John F. DeVine, President, DeVine Laboratories; Dr. William J. Lee, School of Veterinary Medicine, University of Pennsylvania; and Dr. Edward A. Cahill, President, Allied Laboratories, Chicago, Ill.

ASSOCIATION MEETINGS

TWIN CITY VETERINARY MEDICAL ASSOCIATION

After a period of quiescence for more than ten years, the Twin City Veterinary Medical Association, last November, launched another period of activity. The membership is made up particularly of the veterinarians in the Greater Twin City area, who, when counted, number approximately ninety. Their activities in the field of veterinary medicine are many and represent practitioners, the federal meat inspection service, federal and state tuberculosis eradication activities, state live stock disease control activities, state and city public health activities, live stock weighing and inspection service, in the production and distribution of biological products and in teaching and research activities. The place of meeting is the Veterinary Building at University Farm; the date, the third Thursday of the month; and the time is eight p. m.

The first meeting was addressed by Dr. A. E. Wight, chief of the Tuberculosis Eradication Division, U. S. Bureau of Animal Industry. The discussion pertained to the activities of his department in connection with bovine and porcine tuberculosis. Avian tuberculosis was discussed by Dr. W. A. Billings, of the Agricultural Extension Service of the University of Minnesota. A clinic followed which was designed to demonstrate the injection of avian tuberculin, the reactions resulting, the physical appearance of tuberculous birds, and the findings at necropsy.

At the December meeting Dr. G. E. Totten, inspector-in-charge of the meat inspection service at South St. Paul, gave a very interesting talk on this branch of federal government activities. The topic included the meat inspection regulations and their interpretation. Following this, Dr. C. E. Cotton, secretary and executive officer of the Minnesota State Live Stock Sanitary Board, presented some of the facts pertinent to laws and regulations affecting the live stock and veterinary interests of the State.

Dr. C. P. Fitch, chief of the Division of Veterinary Medicine, University of Minnesota, addressed the Association at its February meeting on the subject, "Newer Aspects in the Field of Immunology." He first gave a résumé of the older theories of immunity and followed with a discussion of the newer enzymic conception of immunity and the parts played by the reticulo-

endothelial system of the body. The topic was well illustrated by lantern-slides and graphs.

The March meeting was devoted to a symposium on canine distemper. Dr. R. Fenstermacher, Assistant Professor of Veterinary Medicine, University of Minnesota, presented a very detailed review of the recent investigations of the English Field Distemper Council. Dr. J. S. Dick, a practitioner of Minneapolis, next discussed the symptoms and diagnosis of canine distemper, and Dr. A. A. Feist, a practitioner of St. Paul, the methods of treatment and of handling distemper patients.

Since the social as well as the business side of a veterinarian's life is important, the wives were invited to banquet with the men on the evening of April 18. At 6:30, sixty-seven sat down to eat in the Party Room of the University Cafeteria. After dinner the women adjourned to the Fire Place Room in the Home Economics Building, where they were entertained, while the men gathered in the Veterinary Building and were addressed by Dr. R. A. Gortner, chief of the Division of Biochemistry, University of Minnesota. The subject of his address was "Biochemistry and Medical Problems." This concerned chiefly some of the newer knowledge of colloidal systems of plants and animals. The speaker pointed out that many of the biological phenomena are involved in colloidal systems and reactions.

H. C. H. KERNKAMP, *Secretary.*

DELAWARE STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Delaware State Veterinary Medical Association was held at the Middletown Hotel, Middletown, February 28, 1929. There were thirty-five veterinarians in attendance and the meeting will go down in the history of the Association as the best one ever held. The following program was carried out:

Dr. H. P. Eves, of Wilmington, delivered the presidential address. Dr. Frank Hare, State Poultry Pathologist, presented the subject of "Leukemia in Domestic Fowls." Dr. E. B. Simonds, B. A. I. Inspector-in-Charge of Tuberculosis Eradication, gave a talk on "The Control of Tuberculosis in Cattle." Dr. T. E. Munce, of Harrisburg, Pa., president of the American Veterinary Medical Association, gave a very forceful address on "The Veterinary Profession."

Dr. C. C. Palmer, of the University of Delaware, read a paper, entitled, "Mycotic Pneumonia in Cattle." Dr. L. T. Giltner, Pathological Division, U. S. Bureau of Animal Industry, Washington, D. C., delivered a paper, entitled, "Anthrax Control." This was very thoroughly discussed by Dr. M. L. Zurkow, of Dover.

An election of officers for the ensuing year resulted as follows: President, Dr. F. P. Ruhl, Milford; secretary, Dr. J. R. Porteus, Smyra; Executive Committee, Dr. A. S. Houchin, Stricklersville, Pa., Dr. H. P. Eves, Wilmington, and Louis Levinson, Middletown.

LOUIS LEVINSON, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

MARCH MEETING

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Academy of Medicine Building, Wednesday evening, March 6, 1929.

Dr. Adolph Eichhorn, Director, Veterinary Department, Lederle Antitoxin Laboratories, who had just returned from Europe and who had received first-hand information relative to the new distemper, vaccine was the speaker of the evening. Dr. Eichhorn explained how Dr. Laidlaw and Major Dunkin had labored for five years before perfecting the present vaccine and its method of producing immunity. Dr. Eichhorn explained that the virus is filtrable and is most concentrated in the spleen of infected animals. The product used is the macerated spleen treated with formalin. The vaccine alone is not sufficient to produce a solid immunity and must be used in conjunction with the virus. The virus is unstable and must be kept in a frozen condition to retain its virulence for any length of time. Dr. Eichhorn stated that the best time to vaccinate puppies was at about three months of age.

Dr. J. E. Crawford, Jr., answered the question as to the best treatment for sarcoptic mange in the cat. Dr. D. A. Yandell and Dr. O. E. McKim presented case reports. Dr. D. H. Roth exhibited a Pekingese showing tetany and this condition was discussed by a number of the veterinarians present. Dr. C. S. Chase and Dr. J. J. DeRonde spoke on the proposed bill to increase the fees for dog licenses. A motion was made by Dr. Chase and seconded by Dr. Crawford that the Association go on

record as being opposed to the bill in its present form and the Secretary was instructed to communicate with the Legislative Committee relative to the bill.

A rising vote of thanks was extended Dr. Eichhorn for his interesting talk.

APRIL MEETING

The April meeting was held at the A. S. P. C. A. Hospital, 24th St. & Avenue A, New York City, Wednesday evening, April 3, 1929.

The minutes of the previous meeting were read and approved.

Mr. Merrill Hitchcock, of the A. S. P. C. A., and Mr. Bevan spoke on the spirit of friendliness which now exists between the A. S. P. C. A. and the members of the Veterinary Medical Association. Dr. Robert S. MacKellar spoke on the Code of Ethics which had been adopted by the American Humane Association and was now before the American Veterinary Medical Association for the same purpose.

Dr. Hermann Kock showed a Pekingese which continually cried and voided urine. Dr. J. B. Engle exhibited a Boston Bull with a nervous complaint. These cases were discussed quite freely by several of the veterinarians present.

The application of Dr. Harry Stemen, of New Rochelle, was passed and he was made a member of the Association.

Stereopticon pictures of fractures and foreign bodies, as well as moving pictures of operations were shown by Dr. Raymond J. Garbutt.

A motion was made by Dr. R. W. Gannett and seconded by Dr. Crawford that the Secretary be instructed to correspond with the President of the Jockey Club, relative to the illegal practice of veterinary medicine.

A rising vote of thanks was extended the A. S. P. C. A. for its hospitality. Refreshments were served after the meeting.

RAYMOND J. GARBUTT, *Secretary*

LINN COUNTY VETERINARY ASSOCIATION

At the regular monthly meeting of the Linn County Veterinary Association, held in Cedar Rapids, Iowa, March 8, 1929, the twenty-eight veterinarians present discussed feeds and feeding for two hours and a half. Mr. B. N. Stephenson, Linn County Agent, was the principal speaker of the evening. Mr. S. Lysle Duncan, Johnson County Agent, was present as the guest of

Dr. F. J. Crow, of Iowa City, and Mr. Emmett D. Garner, Cedar County Agent, was in attendance as the guest of Dr. C. H. Banks, of Tipton, Iowa.

The relation of feeds and feeding to animal diseases was discussed at considerable length. Mr. Stephenson gave it as his opinion that a great many of the problems involved in animal nutrition would be solved if the necessary elements, such as minerals and vitamins, were provided in the form of home-grown legumes.

NORTHEASTERN PENNSYLVANIA VETERINARY MEDICAL CLUB

A meeting of the Northeastern Pennsylvania Veterinary Medical Club was held at the Terminal Hotel, Wilkes-Barre, Pa., March 28, 1929. Dr. R. S. Amadon, Professor of Physiology, Veterinary School, University of Pennsylvania, gave an address and demonstration on "The Physiology of Ruminant Digestion." Dr. Amadon illustrated his remarks with lantern-slides, showing phases of his investigations of the subject on live animals at the University of Pennsylvania.

The veterinarians also had the pleasure of listening to Dr. H. W. Turner, sheep specialist of the Pennsylvania Bureau of Animal Industry. Others to address the meeting included Dr. J. J. Thomas, Meat Inspector, Bureau of Animal Industry, and Dr. H. R. Church, Deputy State Veterinarian. All of the addresses were very instructive. The inefficiency of municipal meat inspection as conducted in different cities was discussed from several angles. The attendance numbered twenty. Dr. Thomas D. James, of Scranton, was re-elected Secretary.

McLEAN COUNTY VETERINARY MEDICAL ASSOCIATION

The spring meeting of the McLean County Veterinary Medical Association was held in the Association of Commerce rooms, Bloomington, Ill., March 29, 1929. The meeting was opened at 1:30 p. m. with a symposium on mastitis. Dr. E. H. Marquardt covered the anatomy of the udder; Dr. L. N. Morin, the physiology of the udder; Dr. J. G. Blum, the pathology of udder diseases; and Drs. F. E. Hagy, L. W. Moore and W. A. Smith gave their views on the treatment of mastitis. Dr. L. N. Morin described the operation for amputation of the udder. This

symposium on mastitis developed into a sort of quiz and the members had a lot of fun showing each other how much they had forgotten about their anatomy and physiology. Several said they would know their lesson better the next time.

Dr. W. V. Nesbitt spoke on metritis in the cow and Dr. C. L. Lehman gave his views on calf scours. Dr. A. B. Pletsch was assigned the subject of "Milk Fever."

Dr. Geo. D. Heath, health officer of Bloomington, addressed the meeting on "City Health Service." Dr. Heath has had quite a wide experience in public health work. He congratulated the veterinarians on having sold sanitation and he said that he felt that cities like Bloomington should employ veterinarians as municipal meat and milk inspectors. Of course, the veterinarians present agreed with Dr. Heath.

A short business session followed the program. About twenty members were present and some expressed themselves as believing that this meeting was one of the peppiest ever held. Several said it was the very best.

J. S. KOEN, *Secretary-Treasurer.*

MASSACHUSETTS VETERINARY ASSOCIATION

The annual meeting of the Massachusetts Veterinary Association was held at the Elks Hotel, Boston, April 25, 1929. Fifty members and several visitors were present. A banquet was held, following which the regular meeting took place. As the guest speaker of the evening, the Association was fortunate in having Dr. Geo. H. Bigelow, State Commissioner of Health.

Dr. Bigelow's address was on "The Relation of Diseases of Animals to Humans." In discussing the subject of tuberculosis, Dr. Bigelow stated that there is 50 per cent more infection in rural children, in which no human contact could be traced, than in city children. As comparatively little, if any, milk used on farms is pasteurized, he felt that this explained the fact. Dr. Bigelow said that 25 per cent of the non-pulmonary type of the disease in children is of bovine origin. Referring to Calmette's B. C. G. vaccine, Dr. Bigelow quoted figures to substantiate his statement, that in his opinion the vaccine was not only worthless, but in many instances harmful.

Dr. Bigelow was of the opinion that contagious abortion might spread undulant fever in humans. He based this statement on experiments conducted on some 1200 children in state institutions, 600 of whom received pasteurized milk and were negative

to the agglutination test, whereas the 600 receiving raw milk showed 6 per cent reactors.

The Doctor admitted, however, that several different opinions exist on the titer which indicates a positive reaction. He also felt that there was considerable evidence to show that the porcine strains of *Br. abortus* may be of most significance.

Following Dr. Bigelow's very fine address, various committees gave their annual reports and this was followed by the election of officers for the ensuing year. This resulted in the following officers being elected: Dr. E. A. deVarennes, Quincy, president; Dr. H. F. Dailey, Boston, 1st vice-president; Dr. J. K. Mason, Campello, 2nd vice-president; Dr. H. W. Jakeman, Boston, secretary-treasurer.

A presentation of $100.00 in gold was made to the Secretary-Treasurer in appreciation of his efforts in behalf of the Association during the past five years.

H. W. JAKEMAN, *Secretary-Treasurer*

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The spring meeting of the Michigan-Ohio Veterinary Medical Association was held at Adrian, Mich., April 25, 1929. Veterinarians were in attendance from both sides of the Michigan-Ohio line. Dr. J. M. Timms, of Hudson, Mich., presided. Dr. E. C. W. Schubel, of Blissfield, Mich., presented a very interesting paper on indigestion in horses. It was very freely discussed by all the members present.

An election of officers for the ensuing year resulted as follows: President, Dr. C. H. Hoffmire, Adrian, Mich.; vice-president, Dr. J. H. Lenfestey, Lyons, Ohio; secretary-treasurer, Dr. E. C. W. Schubel, Blissfield, Mich.

E. C. W. SCHUBEL, *Secretary.*

Errata

Dr. Hubert Bunyea wishes to direct attention to two errors in the manuscript of his paper, "A Survey of Poultry Pathology, Past—Present—Future," published in the March, 1929 issue of the JOURNAL.

Page 464, line 22—"inoculated" should be "incubated."

Page 465, line 23—"1925" should be "1825."

NECROLOGY

LEVI P. BEECHY

Dr. Levi P. Beechy, of Columbus, Ohio, died at his home, April 15, 1929, the result of cancer. He was 60 years of age.

The deceased was a graduate of the Ontario Veterinary College, class of 1894. He entered the service of the U. S. Bureau of Animal Industry, April 15, 1898, and was assigned to the meat inspection force at Omaha, Nebraska. Two years later he was transferred to field work, principally in connection with sheep scabies and this assignment took him to a number of the western states. On January 1, 1907, he was transferred to Columbus, Ohio, as Inspector-in-Charge. In 1914, he accepted an assignment to hog cholera control work and continued in this capacity until his retirement, October 1, 1928.

During the 1908 outbreak of foot-and-mouth disease, Dr. Beechy was in charge of eradication work in Ohio. During the 1914 outbreak, he served in Maryland as well as in Ohio. He also saw similar service in California, when foot-and-mouth disease gained an entrance to that state in 1924, and likewise in Texas the following year.

Dr. Beechy was a member of the Ohio State Veterinary Medical Association. He was a Mason and an elder in the Oakland Park Presbyterian Church at Columbus at the time of his death. Dr. Beechy had the respect and esteem of the veterinary profession of the State. He ranked high for his professional training and skill. He leaves a large host of friends in Ohio. He is survived by his widow, one son and one daughter.

A. J. DeF.

JAMES DRURY

Dr. James Drury, of Ypsilanti, Mich., died at his home, March 26, 1929. He had been in poor health for quite a while.

Born in Yorkshire, England, Dr. Drury received his veterinary education at the Ontario Veterinary College and was graduated in 1891. He located in Ypsilanti in 1894 where he continued in active practice until poor health compelled him to retire.

Dr. Drury joined the A. V. M. A. in 1915. He was a member of the Michigan State Veterinary Medical Association. He is

survived by his widow, three sisters and one brother. Burial was at Newmarket, Ontario.

JOHN A. HUHNE

Dr. John A. Huhne, of Kingston, N. Y., died August 9, 1928. He was a graduate of the American Veterinary College, class of 1889. For ten years or more following his graduation, he practiced at Rondout, N. Y., later going to Kingston.

Dr. Huhne was a member of the U. S. V. M. A. from 1890 until 1899.

ROBERT WEIR

Dr. Robert Weir, of Rutland, Vt., died January 27, 1929, from heart failure following an attack of influenza. He was a graduate of the American Veterinary College, class of 1886, and practiced in Rutland for 43 years. For a number of years he was president of the Vermont State Board of Veterinary Medical Examiners.

Dr. Weir joined the A. V. M. A. in 1904.

CHARLES R. SIMPSON

Dr. Charles Rowland Simpson, of Hollywood, Calif., died at his home, April 27, 1929, in his 71st year.

Born in Missouri, Dr. Simpson received his early education in St. Louis. He then removed to Worcester, Mass., where he was employed as an assistant by the firm of J. Penniman and Son, veterinarians. He entered the Montreal Veterinary College and was graduated in 1887. He started in practice at Greenfield, Mass., and the same year removed to Somerville, where he continued in active practice until 1914, when he retired and removed to California.

Dr. Simpson joined the A. V. M. A. in 1901. He leaves two brothers, Rev. L. R. Simpson, of Corder, Mo., and Dr. W. M. Simpson (Montreal '89-McGill '90), of Malden, Mass.

MARCUS J. RAGLAND

Dr. M. J. Ragland died at his home in Salisbury, N. C., May 9, 1929, after an illness of several months, due to complications following an attack of influenza some time ago.

Born in Chattooga County, Ga., October 3, 1878, Dr. Ragland attended public schools of his county and was graduated from Gaylesville College in 1898. He obtained his veterinary training

at the Cincinnati Veterinary College and was graduated in 1905. He entered the service of the Bureau of Animal Industry the same year and was assigned to tick eradication in North Carolina. He resigned from the Bureau in 1909 to engage in private practice at Salisbury. He enjoyed a large practice throughout the years and up until within a few months of his death. Since 1920 he had been veterinarian for the Southern Railroad and for a number of years past he had been water bacteriologist and meat and milk inspector for the City of Salisbury.

Dr. Ragland joined the A. V. M. A. in 1907 and served as resident secretary for North Carolina 1911-1912. He was a member of the North Carolina Veterinary Medical Association from 1909 until his death, served the Association as secretary for the year 1911-1912 and was elected president in 1913. He was a member of the North Carolina State Board of Veterinary Medical Examiners from 1918 to 1928. He also held membership in the American Public Health Association. He was a Mason, a member of Fulton Lodge No. 99, Salisbury Chapter, Royal Arch Masons, Salisbury Commandery of Knights Templar and a Shriner. He also belonged to the Elks and Junior Order.

Dr. Ragland was one of the outstanding veterinarians of North Carolina and a professional man of high rank. He was popular among a large circle of friends and acquaintances. He was a most agreeable man and a genial and companionable fellow. His death is a real loss to his many friends and to the profession. He is survived by his widow and one child, and two children by a former marriage, his mother, one brother and two sisters.

W. M.

MRS. LUCY JONES PEARSON

Mrs. Lucy Jones Pearson died at the home of her daughter, Mrs. William F. Hunt, New Brighton, Staten Island, N. Y., May 19, 1929, at the age of 96, after a short illness.

Mrs. Pearson was the mother of Dr. Raymond A. Pearson, president of the University of Maryland and formerly president of Iowa State College; the late Dr. Leonard Pearson, dean of the University of Pennsylvania School of Veterinary Medicine and state veterinarian of Pennsylvania for many years; the late Edward J. Pearson, president of the New York, New Haven and Hartford Railroad; Mrs. Anne Pearson Warner, of Boston, and Mrs. William F. Hunt. A grandson, Edward Pearson

Warner, was assistant secretary of the Navy in charge of aeronautics under President Coolidge.

It has been the privilege of very few mothers to live and see three sons attain the distinction that hers did in their chosen professions, Raymond in education, Leonard in veterinary medicine, and Edward in engineering. Her two daughters are equally brilliant women. She was married to Mr. Leonard Pearson, general superintendent of the Missouri, Kansas and Texas Railroad, who died in 1884. Before her marriage, Mrs. Pearson taught school in Indianapolis. She educated her children to the point where they were ready for college. She lived for them and with them. For many years she made her home with Leonard in Philadelphia. Later she resided with Raymond, while he was at Ames, Iowa. Immediately prior to her death, she had been living with her daughter Julia. She liked to divide up her time with her children and traveled extensively to do so.

Even at her remarkable age, about a year ago she conceived a notion that she would like to take a ride in an airplane, something that thousands much younger have never had the courage to do. She took the ride and seemed to enjoy it. She retained all her faculties to the last, kept well informed on important developments of the day, was an interesting conversationalist and had her own ideas on many topics. Those who attended the banquet at the A. V. M. A. convention in Philadelphia, in 1927, will remember that Mrs. Pearson honored the occasion with her presence, accompanied by her son, Dr. Raymond Pearson. Truly a most remarkable woman.

PERSONALS

BIRTHS

To Dr. and Mrs. Fred Chandler, of Miners Mills, Pa., a son, Bernard, April 20, 1929.

To Dr. and Mrs. Carl L. Martin, of Durham, N. H., a son, Laurence Alden, May 7, 1929.

To Dr. and Mrs. J. C. Van Ferney, of Scotland, S. Dak., a daughter, Shirley Mae, May 12, 1929.

PERSONALS

Dr. Howard Miller (O. S. U. '06) is a member of the City Council of Columbus, Ohio.

Dr. David W. Cheney (Corn. '22) has removed from Ogdensburg, N. Y., to Bath, N. Y.

Dr. F. Ashton Clarke (A. P. I. '24), formerly of Montgomery, Ala., is now at Andalusia, Ala.

Dr. H. L. Messmore (Chi. '14), formerly of Geneseo, Ill., has located at Oneida, Ill., for general practice.

Dr. J. W. Bryans (Ont. '07), formerly of San Diego, Calif., is now located in Ocean Beach, Calif. Address: 4896 Voltaire Ave.

Dr. Theo. A. Newlin (K. S. A. C. '28), formerly of Lewis, Kansas, is now at the Dixie National Stock Yards, Memphis, Tenn.

Dr. O. F. West (Chi. '11), of Sheldon, Ill., lost the second finger of his right hand as a result of being kicked by a horse in April.

Dr. Mac. L. McLaughlin (Iowa '15), formerly of Los Angeles, Calif., gives a new address, 4280 Campbell Drive, Culver City, Calif.

Dr. D. M. Palmer (Chi. '09), of Ottawa, Ill., has been appointed City Quarantine Officer and entered upon his new duties May 1.

Dr. C. A. Mack (Ont. '01—McK. '02) formerly of Forest Lake, Minn., is now located at St. Paul, Minn. Address: 1267 Seminary Ave.

Dr. A. C. Merrick (O. S. U. '24), formerly of Columbus, Ohio, has located at West Jefferson, Ohio, and will engage in general practice there.

Dr. E. R. Swim (McK. '05) has removed from Altamont, Ill., to Taylorville, Ill., in order that he may live with his mother at the latter place.

Dr. R. W. Grossman (McK. '13), of Columbia, Ill., recently announced his candidacy for the office of County Commissioner of Monroe County.

Dr. C. J. Scott (Iowa '08), the live-wire secretary of the Iowa Veterinary Medical Association, was recently honored by being elected Mayor of Knoxville, Iowa.

Dr. G. W. Northstrum (McK. '10), of Crystal Falls, Mich., was a patient at the Mayo Clinic, Rochester, Minn., during April. He went there for an operation for goiter.

Dr. G E. Keith (Ind. '18), who has been employed by the Indiana Live Stock Sanitary Board for the past ten years, has located at Plymouth, Ind., for general practice.

Dr. Norman Hanson (O. S. U. '20) has completed his postgraduate work at the University of Pennsylvania and is now in Minneapolis. Address: 1111 Sixteenth Ave. S. E.

Dr. John B. Cheney (Corn. '24), who has been located at Potsdam, N. Y., has accepted a position with Borden's Farm Products Company, and will be stationed at Oxford, N. Y.

Dr. W. L. Williams (Mont. 79), of Ithaca, N. Y., has decided to go to the Territory of Hawaii for an indefinite stay. He has requested that his mail be addressed to him at Kamuela, Hawaii

Dr. D. M. Swinehart (O. S. U. '10), who has been a field veterinarian for the Department of Agriculture of Ohio for a good many years, has left the service and has located at Elida, Ohio, for general practice.

Dr. C. H. Hoffmire (O. S. U. '08), who practiced for seven years at Continental, Ohio, has removed to Adrian, Mich., where he has taken over the practice of the late Dr. George D. Gibson (Ont. '93).

Dr. M. A. Emmerson (Iowa '25), instructor in the Department of Anatomy, Iowa State College, is planning to spend the school year 1929-1930 in post-graduate study at the University of Zurich, Switzerland.

Dr. Dea McDonald (Ind. '11), of Kendallville, Ind., has been employed by the Bayer Ice Cream Company, of Kendallville, to inspect the herds of all farmers selling milk to the Bayer Ice Cream Company plant.

Dr. O. L. Sutton (Cin. '11) has moved into his new quarters at 3112 Fairfield Avenue, Walnut Hills, Cincinnati, Ohio, where he has a large lot with a two-story building in the rear, which he will use for hospital purposes.

Dr. O. C. Anderson (McK. '15), who has been associated with Dr. E. B. Cavell (Ont. '06), of Northville, Mich., for several years, has returned to Detroit and is again associated with Dr. E. G. Folsom (Ont. '08-'20).

Dr. H. T. Farmer (U. S. C. V. S. '11), has located at 3100 North Avenue, Richmond, Va., following the dissolution of the firm of Farmer and Rowe, who conducted the Fraser Veterinary Hospital, of Richmond, Va., for a number of years.

Dr. M. W. Ray (K. C. V. C. '11), who has been state veterinarian of South Dakota for about four years, has removed from Pierre to Sioux Falls, where he will represent the Corn States Serum Company, of Omaha, Nebr., and the Haver-Glover Laboratories, of Kansas City, Mo.

Dr. E. H. Durr (A. P. I. '24) has resigned his position as Assistant State Veterinarian of North Carolina and has begun work with the Mississippi State Live Stock Sanitary Board, having been assigned to infectious abortion control work. His headquarters are at A. & M. College, Miss.

Dr. F. A. Zimmer (O. S. U. '09), former state veterinarian of Ohio, has returned to Pataskala and will engage in general practice there. Dr. Zimmer recently completed the construction of a veterinary hospital, containing an office, an operating room, a laboratory and two sick wards.

Dr. T. A. Sigler (Ind. '02), of Greencastle, Indiana, presented a beautiful cemetery arch to the Clinton Falls M. P. Church, Sunday, May 12, 1929, in connection with a Mother's Day program. The arch is of brick, with stone tablets, and is a beautiful structure from the architectural standpoint.

Dr. Alan Bachrach (U. P. '28), of Philadelphia, Pa., has been up in the wilds of Canada for almost a year in charge of the wild animals belonging to Burden Pictures, Inc. His organization has been making a picture at Fabre, Que., and Dr. Bachrach expects to return to his home in Philadelphia in the near future.

Dr. J. M. Jones (McK. '12), of Lewisburg, Tenn., has been appointed State Veterinarian by Governor Horton, to succeed Dr. W. B. Lincoln (Iowa '93), who has served as Tennessee's chief veterinary control official for several years. Dr. Lincoln was appointed during the first term of Governor Peay. The change is effective June 1.

Dr. F. L. Roach (Chi. '04), of Preston, Iowa, completed twenty-five years of practice on May 16, 1929. During this time Dr. Roach served his city as Mayor and as member of the School Board for several terms. He is president of the Jackson County (Iowa) Veterinary Medical Association and a member of the Executive Committee of the Eastern Iowa Veterinary Association.

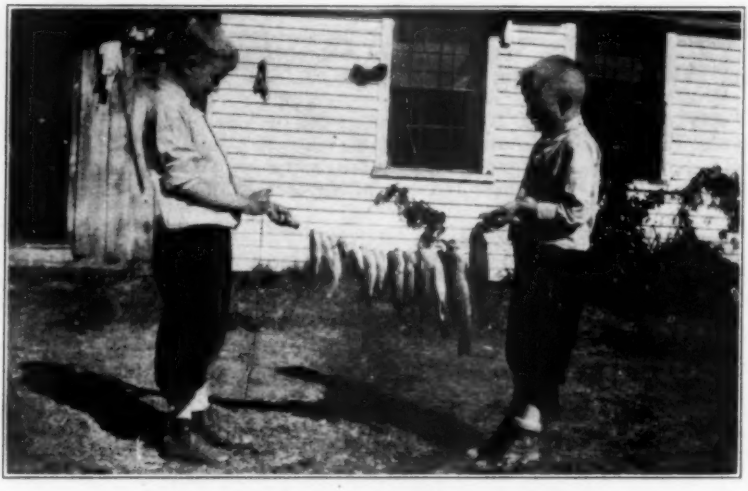
Dr. C. F. Pangburn (Gr. Rap. '04—Ind. '09), of Charlestown, Ind., member of the Indiana Live Stock Sanitary Board, was seriously injured the early part of May, when he was struck by a milk truck on State Highway No. 31, near Underwood. He was removed to a Louisville hospital, where it was found that he had suffered internal injuries in addition to a number of fractured ribs.

Dr. T. H. Edwards (K. C. V. C. '09), formerly of the Los Angeles City Health Department, moved to El Paso, Texas, the first of the year, seeking a more congenial climate. He was successful in securing appointment as City Meat Inspector and assumed his new duties on March 1. Dr. Edwards plans to take the State Board examination this month and expects to be able to do some practicing.

Dr. O. V. Brumley (O. S. U. '97), of Columbus, Ohio, secretary of the veterinary faculty at Ohio State University and vice-president of the Ohio State University Athletic Board, has been appointed a member of the Columbus Board of Health for a four-year term by Mayor Thomas. Dr. Brumley enjoys the distinction of being the first veterinarian to be appointed a member of the local board of health.

Dr. C. A. Deadman (Gr. Rap. '03), of Madison, Wis., has been appointed State Veterinarian of Wisconsin, by Commissioner of Agriculture Walter Duffy. Dr. Deadman assumed his new duties April 6. Incidentally this was his birthday. The new position will not prevent Dr. Deadman from continuing to act as a member of the Common Council of the City of Madison. He also took his oath of office as councilman on April 6.

Dr. G. A. Roberts (K. C. V. C. '03), who has been on leave of absence from the Institute Evangelico, Escola Agricola de Lavras, Lavras, Minas, Brazil, for the past year, and who has been at the Polytechnic Institute at San German, Porto Rico, for about five months, has been asked to go to the Experiment Station of the Virgin Islands, on the island of St. Croix, near Christiansted. Dr. Roberts will have the title of Veterinarian and Animal Husbandman while at the Station.



TEACHING THE YOUNG IDEA HOW TO FISH

Sons of Dr. Donald B. Palmer, of Minneapolis, Minn., with fish caught near Muskegon, Mich., in the month of August. The small fish are rock bass (*Ambloplites rupestris.*) The large ones are small-mouthed black bass (*Micropterus dolomieu.*)

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